

# South African Medical Journal

Organ of the Medical Association of South Africa



# S.-A. Tydskrif vir Geneeskunde

Vakblad van die Mediese Vereniging van Suid-Afrika

Incorporating the South African Medical Record and the Medical Journal of South Africa

REGISTERED AT THE GENERAL POST OFFICE AS A NEWSPAPER

Vol. 26, No. 17

Cape Town, 26 April 1952

Weekly 2s 6d

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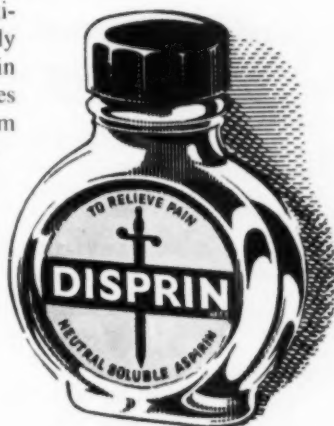
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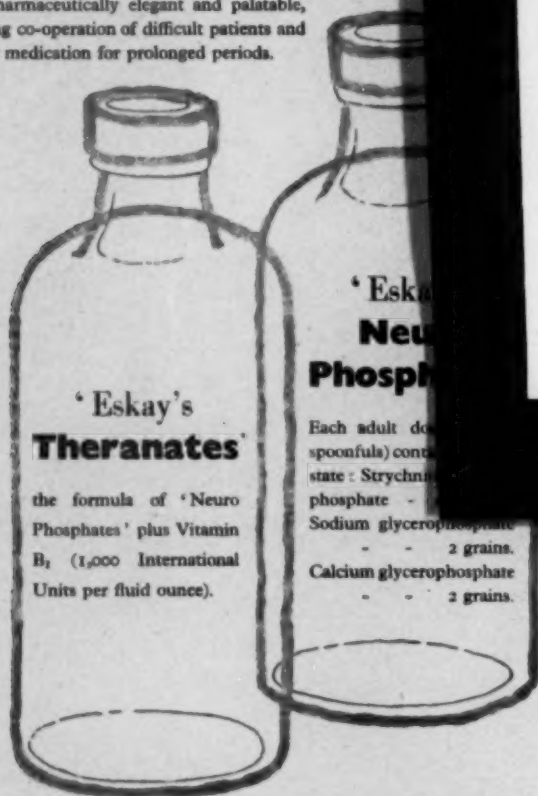
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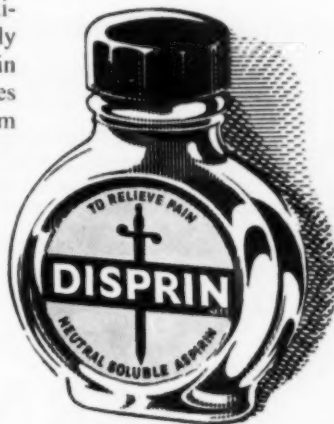
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# South African Medical Journal

## Suid-Afrikaanse Tydskrif vir Geneeskunde

P.O. Box 643, Cape Town      Posbus 643, Kaapstad

Vol. 26, No. 17

Cape Town, 26 April 1952

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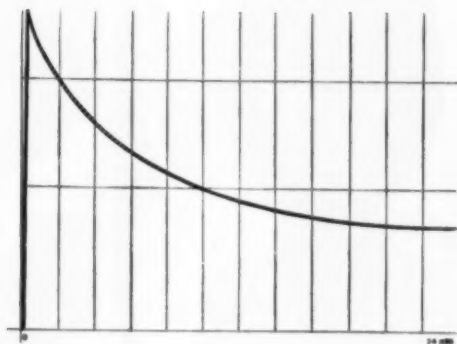
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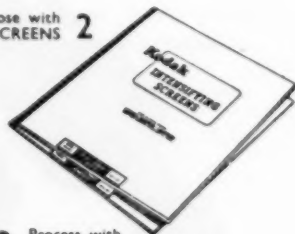
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# South African Medical Journal

## Suid-Afrikaanse Tydskrif vir Geneeskunde

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Vol. 26, No. 17

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### THE PROTHROMBIN LEVEL IN PATIENTS ON DICOUMAROL THERAPY

#### A SIMPLIFIED CAPILLARY TECHNIQUE

H. B. STEIN, M.Sc., M.D., D.P.H. (RAND), D.C.P. (LOND.)

and

B. V. WALLACE, B.Sc. (S.A.), M.B., B.Ch. (RAND)

Department of Clinical Pathology, University of the Witwatersrand

This communication presents a study made on the control of Dicoumarol therapy by bedside prothrombin determinations, using a simplified technique capable of being carried out by the general medical practitioner. We became interested in the possibilities of the method when we were requested by country practitioners with limited laboratory facilities to assist them in the control of serious cases at their homes. It might have proved dangerous to transport these cases to urban hospitals, as they suffered mainly from coronary thrombosis and thrombo-embolic phenomena.

We do not recommend that the technique to be described should supplant the standard laboratory methods recommended for prothrombin determinations. We have used the modification of Quick's one-stage method described by Stein<sup>1</sup> (hereinafter referred to as the 'standard' technique) on almost all cases undergoing anticoagulant therapy with satisfactory results. However, in cases of emergency, where inadequate laboratory facilities exist, the method described below (referred to as the 'capillary' technique) may prove of value in controlling the dosage of Dicoumarol.

#### METHOD

The capillary method is not new. It is a modification of methods described originally by Quick<sup>2</sup> and Kato,<sup>3</sup> and used heretofore only in infants.

**Thromboplastin Extract.** This is prepared from human brain obtained in as fresh a state as possible at autopsy. The vessels and pia mater are removed with forceps, the process being facilitated by washing under running water from a tap. The cortex is separated as completely as possible from the medulla, since the cortex appears to provide a more satisfactory extract and is more easily dried than the medulla. The selected brain portions are then ground in a mortar with a little acetone for about 3-4 minutes, the acetone is discarded, fresh acetone is added and the grinding process repeated. After a few washings with acetone the brain powder can be finely ground into an almost dry powder. Further drying of the final acetone wash is achieved by using a water suction filter and drying in an evacuated desiccator containing calcium chloride.

The dried powder is stored in rubber-capped 1-oz. bottles and air in the bottle is evacuated with a small electric pump attached to a needle which pierces the cap. The seal is covered with molten paraffin wax which is allowed to cool. The

bottles are stored in the refrigerator at 4°C. After each removal of powder, the bottle is re-evacuated and re-sealed. This method of preparing the brain powder is essentially that of Aggeler *et al.*<sup>4</sup>; it furnishes the thromboplastin extract which we prepare in the laboratory for daily use with the standard technique and replaces the dried brain powder previously used.<sup>5</sup>

For the capillary test, a fresh 6% suspension of the brain powder is prepared in normal saline at 45°C. The suspension is placed in a 37°C incubator, mixing the contents every 5 minutes. After 30 minutes the suspension is centrifuged. The milky supernatant fluid is removed and pipetted in 0.25 ml. quantities into a series of ampoules of 2 ml. capacity. The latter are frozen by immersion in carbon dioxide snow and evacuated by means of a Speedivac high-vacuum pump while frozen. After freeze-drying, the ampoules are filled with dry sterile nitrogen and sealed. The resultant dried thromboplastin will now last for at least several months (some of our batches have lasted 9 months) and should preferably be stored in the refrigerator. The thromboplastin extract which one requires for the test is then simply prepared by opening the ampoule and adding 0.25 ml. of distilled water to dissolve the freeze-dried powder.

Several measuring pipettes to deliver 0.02 ml. are prepared from Pasteur pipettes whose ends are rounded off in a small flame. A volume of mercury corresponding to 0.02 ml. is drawn up into each pipette. The upper level is marked with hydrofluoric acid and then gone over with India ink. Such pipettes can be cleaned, dried and used repeatedly. They do not break if carefully handled. They replace the haemoglobinometer pipettes recommended by Kato,<sup>3</sup> and are much more economical.

For the test 0.02 ml. of the reconstituted extract is first placed on a clean microscopic slide. The finger of the person being tested is cleaned with ether and pricked with a Hagedorn needle; 0.02 ml. of capillary blood is sucked up in a clean Pasteur pipette, avoiding excessive squeezing of the wound to minimize the amount of tissue extract expressed. The blood is placed on the slide next to the drop of thromboplastin extract. The blood and extract are mixed with a clean steel pin and a stop-watch started. An ordinary watch with a second-hand will do if no stop-watch is available. The point of the pin is passed repeatedly through the blood and extract mixture, and the onset of clotting noted by the first appearance of fibrin which adheres to the pin. This clotting time is the 'prothrombin time'. A normal control prothrombin time (from the doctor himself or a relative of the patient) is performed similarly. The patient's 'prothrombin index %' is then calculated as follows:—

$$\frac{\text{Prothrombin Time of Normal Control}}{\text{Prothrombin Time of Patient}} \times 100.$$

Different batches of extract have given prothrombin times in normal individuals of 25 to 35 seconds by the capillary method. The average normal prothrombin time for each batch is indicated when originally tested, and the practitioner is advised not to use the extract when the time is prolonged more than 15 seconds beyond the stated period.

The freshly prepared ampoule provides enough material for about 10 tests. Although the material will last for nearly a week if kept in the refrigerator, we recommend that a fresh ampoule be used daily. The doctor to whom the ampoules are sent is requested to familiarize himself with the technique by testing himself and other normal persons with one ampoule to confirm the approximate normal prothrombin time of the batch.

If properly dried and sealed *in vacuo* the powder maintains its white colour and forms a milky white extract on addition of the water, but if the powder yellows or the extract produced on adding water is yellow, the ampoule has not been properly prepared and will give prolonged prothrombin times. Such yellow extracts should be discarded.

#### RESULTS

Different batches of brain have been tried with success. These batches have given excellent correlations with extracts used in the laboratory in the standard technique.

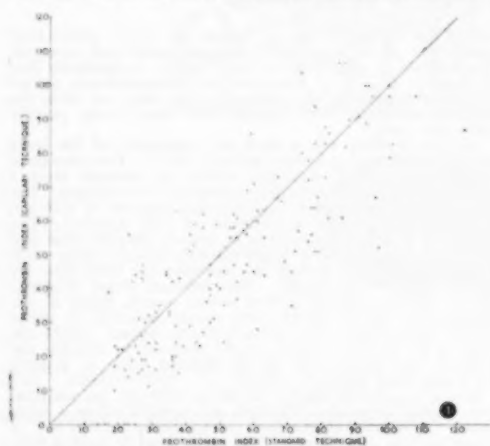


Fig. 1. Correlation of Standard and Capillary Techniques. Coefficient of Correlation: 0.82.

In order to compare the prothrombin indices employing the same brain in the capillary (using the freeze-dried ampoule) and standard (using the powder made daily as 6% suspension in normal saline) methods, 200 determinations were made on patients under treatment with Dicoumarol, both methods being used simultaneously to estimate the prothrombin index. The results are indicated in Fig. 1. The over-all coefficient of correlation was found to be 0.82. The correlation holds best when the prothrombin index lies above 50%. With lower levels the correlation is less, the prothrombin index by the standard laboratory method tending to be higher than by the capillary method. This is no disadvantage, since the capillary method will thus be weighted for safety by tending to prevent the lower levels being reached by the standard method.

#### DISCUSSION

The mechanism of Dicoumarol action is at present incompletely understood. Occasionally, on addition of the usual amount of thromboplastin, the plasma of a Dicoumarolized patient may actually be prevented from clotting, instead of the usual accelerating effect of the thromboplastin on coagulation becoming manifest. We have observed a case which was given 1,300 mg. Dicoumarol over a few days without prothrombin levels being determined, and who bled per vaginam. On admission to hospital the prothrombin time by the standard method could not be determined as the plasma failed to clot in 10 minutes. However, on testing the recalcification time of the plasma (i.e. without thromboplastin extract), the plasma was observed to clot in 4 minutes. A similar anomalous finding has been observed experimentally in dogs poisoned with Dicoumarol (Bingham *et al.*<sup>3</sup>), while Witts<sup>12</sup> and Macfarlane<sup>7</sup> have also observed a clinical case somewhat analogous to ours. Furthermore, Witts has observed quite different prothrombin times of dicoumarolized patients when using brain extracts from different species. Again the same plasmas will give divergent results when Russell viper venom is used as a source of thromboplastin as compared with brain extract (Witts<sup>12</sup>; Biggs and Macfarlane<sup>2</sup>; James<sup>5</sup>). For the control of Dicoumarol therapy it would appear that brain extracts of the same species being tested provide the best means of assessment.

Despite the fact that the action of Dicoumarol on blood coagulation has not been clarified, there is no doubt that the method for estimating prothrombin time using human brain extract provides a suitable method of laboratory control of Dicoumarol therapy. It is recommended that the original prothrombin time of the patient (which, in the absence of diseases causing hypoprothrombinaemia, may be assumed to be normal), whether determined by the capillary or standard method, be prolonged 2 to 2½ times to maintain an effective therapeutic level for the prevention of thrombo-embolic phenomena. This corresponds to a prothrombin index of 40-50%.

We still prefer to record our results as an index. It ensures the use of a normal control with each test and is in any case the only means of expressing the result in the capillary technique described in this paper. The use of the term 'prothrombin concentration' is at best pseudo-scientific, especially as we do not know the chemical constitution of prothrombin and as a biological clotting technique is used to estimate the results. Should, however, one desire to convert the 'prothrombin index' to 'prothrombin concentration', one may readily do so by reading off the results on curves constructed in the paper by Stein (1941, Fig. 1).<sup>9</sup>

One wonders whether the fatal haemorrhagic complications recorded in America and elsewhere (Duff and Shull<sup>4</sup>) are not due to the use of animal extracts and the employment of the 'prothrombin concentration' method in recording results. The 'prothrombin index' curve is a smooth one on which a 5% difference is the same anywhere along the curve. In contrast, the 'prothrombin concentration' is a sharply tapering curve, so that 5% difference between 12% and 7% concentration is very marked indeed; while 12% concentration is just within the therapeutic level, 7% is very close to the danger region.



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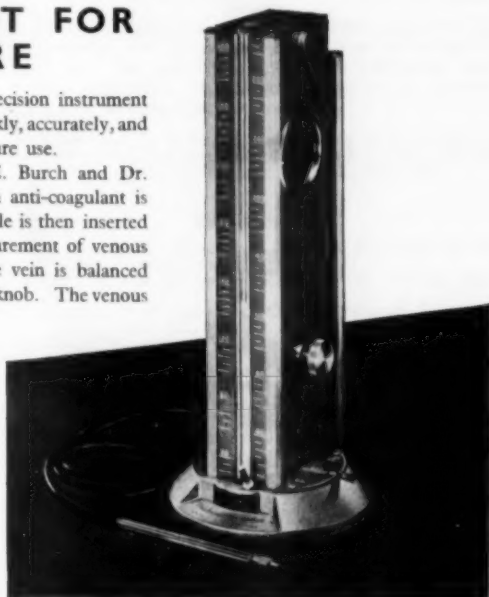
The simple technique used is that devised by Dr. George E. Burch and Dr. Travis Winsor of the Tulane University Medical School. An anti-coagulant is drawn up into the needle and observation tube and the needle is then inserted into the vein. A small gauge needle is used, permitting measurement of venous pressure in small as well as large veins. The pressure in the vein is balanced manually with the PHLEBAUMANOMETER pressure control knob. The venous pressure in millimeters of water is read directly from the graduated scale.

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The non-mathematical mind tends to regard these two levels (7% and 12% concentration) as being not very different and this may be one of the reasons for over-dosage. Until prothrombin can be estimated by chemical means, we prefer to adhere to the use of the term 'prothrombin index %' in expressing the prothrombin level in clinical cases.

The capillary technique described above can, we feel, be used effectively for controlling Dicoumarol therapy in emergency cases with limited laboratory facilities. We do not recommend it to supplant the standard technique where adequate laboratory facilities exist, since with the standard technique the temperature of the test is controlled and several determinations can be made on the same sample of plasma. In other words, we still stand by the remarks made by Stein in 1941,<sup>9</sup> where bedside procedures were condemned. However, in emergency cases the capillary method described here may prove of definite value, especially in South Africa with its sparsely populated, widely scattered areas.

Finally, we must stress that the capillary method should not be used for estimating the prothrombin response to vitamin K in the differentiation between intra- and extra-hepatic jaundice,<sup>10,12</sup> since it is too crude for this purpose and will only serve to bring this useful diagnostic test into disrepute.

## SUMMARY

A simplified capillary method for the determination of the prothrombin level in patients on Dicoumarol therapy is described. The use of the method in emergency cases only is emphasized, and its relationship to the standard laboratory technique is discussed.

Our thanks are due to Dr. A. Zoutendyk, of the South African Institute for Medical Research, who was responsible for the freeze-drying of the powder used in the capillary tests.

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## QUESTIONS ANSWERED

## CREEPING ERUPTION

**Q.** Will you kindly give me some information about etiology and treatment of the following:—

(a) *Builder*. History: Three weeks before he had been on the beach. He developed a very irritating lesion on the side and the sole of the right foot. He described it as 'a thin line 2 inches long with 2 small blisters at the end'. When I saw this patient the foot was swollen and there was an ulcerated patch about 3 inches in diameter on the side and under the foot. No sign of the original lesion was present and the ulcer was in a dirty condition due to cement and road soil.

After treatment with Cetavlon, injections of Penicillin and tar paste followed by Elastoplast dressing, the whole area is completely healed.

(b) *Manager*. He consulted me about a 'ring-worm' on the lower abdominal wall. O.E.: There was a thin inflamed track about 3 inches long, winding in 3 directions. Four blisters were present and these were filled with a thin, watery fluid. The lesion itched intensely. *Not Tinea circinata*. This was treated at first by benzyl benzoate locally, then generally, but the track extended and more irritating blisters appeared.

I used ethyl chloride spray on this case, and after almost daily applications it is now almost healed. This man had been on the same beach as (a) 10 days before.

(c) *Housewife*. She came to see me when 4 lesions were well developed. These have been very resistant. With ethyl chloride spray the tracks were opened up and strong iodine applied for 3 weeks. Ethyl chloride alone failed to arrest the extension of the tracks. All these tracks were opened and iodine applied.

Except for a residual hyperaemia on part of the treated area, these are now healed and no pruritus is now present, but the patient returned to me to-day with 2 fresh tracks on the lower edges of the labia minora and pruritus vulvae.

Do you know what this parasite is? I cannot find anything

in the medical or skin books about it, and the ethyl chloride-iodine treatment is impossible in the last-named area. I have given the patient S.V.C. tablets p.v. to try to prevent an ascending infection. Both cases (b) and (c) are on Hetrazan by mouth but not knowing the etiology of the condition is very unsatisfactory.

I should be very grateful if you or your colleagues could give me some help in these cases. Is there a specific for the condition?

I have been told that the river mouths around East London are the only places where this 'worm' exists. In all my 20 years in general practice and my year in India, I have never met anything like it, although it does slightly resemble the 'guinea worm'.

**A.** The wavy linear and serpiginous tracts described in these cases are undoubtedly due to the larva migrans (creeping eruption or sand-worm disease). The larva is said to be an immature stage of the dog and cat hookworm, *Ankylostoma brasiliense*, or of an allied species. In South Africa it is especially prevalent on the south-east coast. Cases are often seen in the Cape Peninsula.

References to this disease can be found in any modern textbook on dermatology. An excellent description with illustrations is given by Loewenthal in *Modern Practice in Dermatology* (Mitchell-Heaggs).

The usual treatment consists of freezing the advancing edge of the tract with ethyl chloride for 2 minutes. This may have to be repeated 2 or 3 times in some cases. Other local measures include application of tincture of iodine or pure carbolic, or careful injection of bichloride of mercury or chloroform into the tract.

Multiple and wide-spread infestation may require systemic treatment. Hetrazan in full doses is useful. Various antimony compounds are said to give good results.

# South African Medical Journal

## Suid-Afrikaanse Tydskrif vir Geneeskunde

### EDITORIAL

#### MALARIA SURVEY OF SOUTH WEST AFRICA

Towards the end of last year a Malaria Conference was held at Kampala, Uganda. Many important papers were presented and amongst these was a malaria survey of South West Africa by Dr. Botha de Meillon, entomologist on the staff of the South African Institute for Medical Research, Johannesburg. Dr. de Meillon's report has recently been published in the *Bulletin of the World Health Organization*, 1951, Vol. 4, No. 3. He points out that the climate of South West Africa is of the desert type, i.e. hot days followed by very cold nights. The cold Benguela current flowing up the west coast plays an important part in determining the climate. This climate undoubtedly has an influence on malaria endemicity and is thought to play some part in several instances of 'anophelism without malaria', encountered.

The presumed vectors are *Anopheles gambiae* over the whole territory plus *A. funestus* on the Okavango and probably the Kunene. Sufficient dissections could not be undertaken to confirm this definitely. The possible role of *A. listeri* as a vector was not settled. The distribution of *gambiae* has been usefully mapped.

The bionomics and relation to malaria of *A. gambiae*, *A. funestus*, and *A. listeri* are dealt with in some detail.

The endemicity of the disease was estimated from spleen- and parasite-rates of different age-groups of indigenous native people. The varying degrees of endemicity are classified according to Wilson's four groups. Malaria of the highest endemicity (Wilson's Group I) occurs along the Okavango River and probably on the Kunene and its immediate environs. From there south the degree of endemicity gradually decreases and most of the country south of latitude 19° south falls into Wilson's lowest group. This large area is subject to either localized or widespread epidemics.

*Plasmodium falciparum* accounts for 91% of malaria in the areas surveyed. *P. malariae* and *P. vivax* make up the rest in approximately equal proportions.

Although the control and eradication of vectors is not dealt with exhaustively, the view is expressed that the eradication of *A. gambiae* from the greater part of South West Africa does not present insuperable difficulties. Control by means of residual insecticides offers the best hope for the highly endemic regions on the Okavango and in Ovamboland.

### VAN DIE REDAKSIE

#### MALARIA-OPNAME VAN SUIDWES-AFRIKA

Teen die einde van laasjaar is daar 'n Konferensie oor Malaria in Kampala, Oeganda, gehou. Baie belangrike referate was gelewer o.a. 'n malaria-opname van Suidwes-Afrika deur dr. Botha de Meillon, entomoloog van die personeel van die Suid-Afrikaanse Instituut vir Mediese Navorsing, Johannesburg. Dr. de Meillon se verslag is onlangs in die *Bulletin van die Wêreld-Gesondheids-organisasie*, 1951, Vol. 4, No. 3, gepubliseer. Hy wys daarop dat die klimaat van Suidwes-Afrika van die woestyn-tipe is, d.w.s. warm dae wat deur baie koue nagte gevolg word. Die koue Benguela-stroom wat langs die Weskus noordwaarts vloei, speel 'n belangrike rol in die bepaling van die klimaat. Hierdie klimaat het ongetwyfeld betrekking op malaria-endemiteit en dis die mening dat dit te doen het met verskeie gevalle van 'anofelisme sonder malaria' wat teëgekomp is.

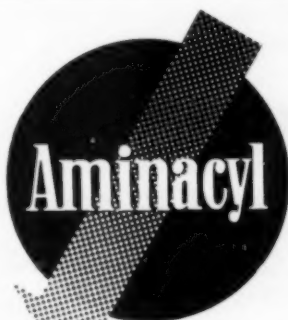
Dit word aanvaar dat *Anopheles gambiae* draers oor die hele gebied is plus *A. funestus* langs die Okavango en moontlik die Kunene. Voldoende disseksies om dit definitief te bevestig, kon nie onderneem word nie. Die moontlike rol van *A. listeri* as 'n draer was nie bepaal nie. Die verspreiding van *gambiae* was gerieflik op die kaart aangetoon.

Die bionomie en verhouding tot malaria van *A. gambiae*, *A. funestus*, en *A. listeri* word ietwat in detail behandel.

Die endemiteit van die siekte was bereken van die milt- en parasiet-sifers van verskillende inheemse inboorlingstamme. Die wisselende grade van endemiteit is volgens Wilson se vier groepe geklassifiseer. Malaria van die hoogste endemiteit (Wilson se Groep I) kom langs die Okavango-rivier voor en moontlik langs die Kunene en sy onmiddellike omgewing. Daarvandaan suid verminder die graad van endemiteit geleidelik en die grootste gedeelte van die landstreek suid van breedtegraad 19° suid val onder Wilson se laagste groep. Hierdie groot gebied is of onderhewig aan plaaslike of wydverspreide epidemies.

*Plasmodium falciparum* is verantwoordelik vir 91% van die malaria in die gebied wat opgeneem is. *P. Malariae* en *P. vivax* maak die res in ongeveer gelyke verhoudings uit.

Hoewel die beheer en uitwissing van draers nie baie omvattend behandel word nie, word die mening gehuldig dat die uitwissing van *A. gambiae* uit die grootste gedeelte van Suidwes-Afrika nie onoorkoomlike moeilikhede inhou nie. Beheer deur middel van oorblywende insektmiddels bied die beste hoop vir die hoë endemiese streke langs die Okavango en in Ovamboland.

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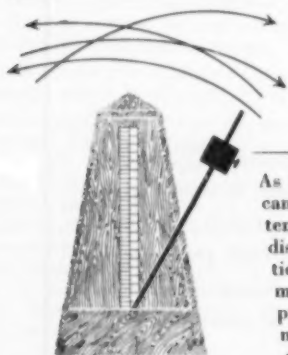
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## FLOATING GALL STONES

## REPORT OF A CASE

W. WILKIE, CH.M.

Cape Town

Eliasz (1931)<sup>1</sup> first drew attention to a horizontal band of translucency produced by non-opaque gall stones floating in a dye-filled gall bladder. He performed the simple experiment of collecting samples of bile by duodenal intubation and pouring these into a test tube, when he found that they did not mix but formed layers—the most concentrated bile forming the lowest layer. He suggested that gall stones that were light enough to float would do so in that layer of bile in which the density equalled their own specific gravity.

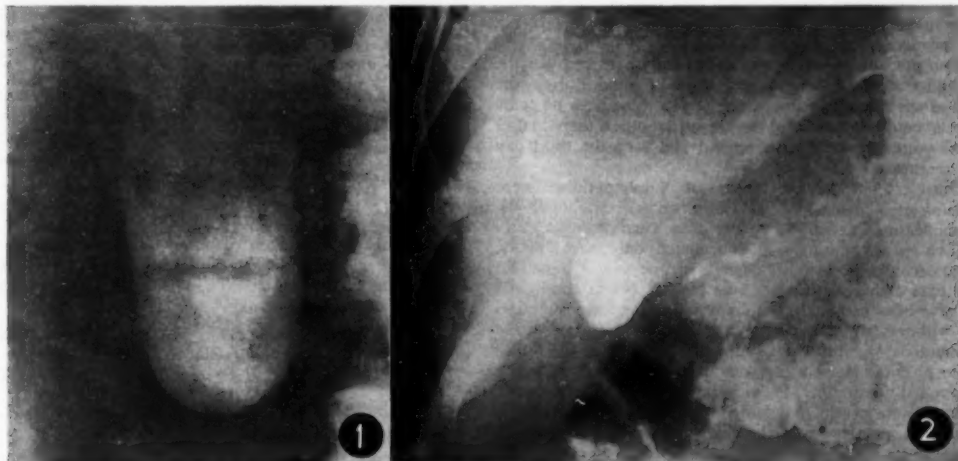
This sign of floating gall stones is well known to-day and is seen not infrequently, but a double layer of floating stones is distinctly rare and as no report of this could be found in the literature it was thought worth recording a case.

The patient, a European male aged 45 years, gave a

duct but producing no obstruction to either filling or emptying of the gall bladder.

At operation, 3 large mixed and numerous small mixed stones were found in the gall bladder and 2 small mixed stones in the cystic duct. The gall bladder showed evidence of chronic cholecystitis in that there was a subserous deposit of fat, the cystic gland was enlarged, and the gall bladder separated from the liver with considerable difficulty.

*Discussion.* The need for at least one view taken in the erect position and preferably with compression is generally accepted to-day in cholecystography, as this is often the only means of demonstrating non-opaque floating stones. Without this view the gall bladder would have been passed as normal, as concentration of dye and also emptying after fatty meal were good.



history of typical attacks of gall-stone colic without jaundice occurring during the last 5 years. Between attacks he was fit and had no dyspepsia. He had been completely free of pain for the last 2 months during which time a cholecystogram was taken. The clinical diagnosis was gall-stone colic with no associated chronic cholecystitis.

The cholecystogram showed a gall bladder that concentrated the dye well, thus confirming the clinical diagnosis of the absence of cholecystitis. In the erect view (Fig. 1) 2 horizontal bands of translucency were seen, the upper one wider than the lower, and obviously produced by three large floating gall stones; no individual stones could be made out in the lower band but it was obviously due to a layer of smaller stones.

The picture taken after a fatty meal (Fig. 2) showed that the gall bladder had contracted well and the cystic duct had filled with dye, in which could be clearly seen 2 circular filling defects, suggesting stones lying in the cystic

duct. Although radiologically and clinically there was no evidence of chronic cholecystitis, yet at operation definite signs of it were present.

Stones are seldom demonstrated radiologically as filling defects in the cystic or common ducts because the dye leaves the duct so rapidly that they are practically always empty when the exposure after the fatty meal is made. In this case the stones in the cystic duct, although not obstructing it completely, did produce enough stasis to allow visualization of the cystic duct.

*Summary.* A case is presented with a cholecystogram showing a double layer of floating stones and filling defects in the cystic duct due to stones.

I should like to thank Dr. M. Zion for referring this case to me.

## REFERENCE

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## BACTERIAL PNEUMONIA

W. F. SCOTT, M.D., B.Ch. (RAND), M.R.C.P.

Coronation Hospital, Johannesburg

(Concluded from p. 339)

## CHEMOTHERAPY

In the series of 52 cases, Sulphapyridine was used in all cases treated chemotherapeutically. The drug was commenced almost immediately after the diagnosis of pneumonia was made, with an initial dose of 2 gm. followed by 1 gm. 4-hourly. The average dose was 28 gm., the smallest dose was 14 gm., and the largest 68 gm. One case with a lesion involving the whole of the right apical lobe did not respond, but responded rapidly to 100,000 units of Penicillin by intramuscular injection.

Criticism may be levelled at the use of Sulphapyridine when less toxic newer sulphonamides are available but, as Sulphapyridine was standard treatment in the hospital, these cases were treated with the same drug to preserve uniformity.

One precaution which was consistently enforced was the administration of liberal quantities of fluid during the course of Sulphapyridine therapy. In this series no toxic effects could be ascribed to the drug other than that 3 cases vomited, one on the second and 2 on the third day of treatment.

During 1948, 98,000 tablets of Sulphapyridine were administered in the medical wards for various complaints. Complications ascribable to the drug were rare; the commonest was vomiting which occurred only occasionally. Apart from this, in this hospital, in one year I have had one case of haematuria and one case of anuria considered to have been caused by Sulphapyridine; the former cleared on withdrawal of the drug and the latter passed urine spontaneously within 24 hours of withdrawal. In other mine hospitals I have had one case of haematuria which responded favourably to withdrawal of the drug, and a case of anuria which persisted for 5 days—irrigation of the renal pelvis afforded relief with passage of urine. These 4 cases were recorded over a period of 18 months.

## BACTERIOLOGY

Thirteen reports on the bacteriology of the sputum from the series were made available and demonstrated the organisms to have occurred with the following frequency:—

1. <i>Micrococcus catarrhalis</i> ... ..	8
2. Non-haemolytic streptococcus ... ..	6
3. <i>Pneumococcus</i> ... ..	5
4. <i>B. Friedlander</i> ... ..	4
5. <i>H. influenzae</i> ... ..	3
6. Haemolytic streptococcus ... ..	1
7. <i>Staphylococcus albus</i> ... ..	1
8. <i>B. proteus</i> ... ..	1
9. Yeast ... ..	1

The cases seen were thus associated with a mixture of organisms in the sputum, except for one case in which a pure culture of *Staphylococcus albus* was isolated. The only reliable method of determining which particular organism is responsible for the pneumonia would be to puncture the lung and draw

off organisms from the consolidated area, a difficult and impracticable procedure in cases with small lesions.

With the object of determining the bacterial flora present at the time, Ordman<sup>16</sup>, commencing in November 1948, carried out a 2-month bacteriological survey of the sputa collected from cases of pneumonia among Native mine labourers employed on the various mines on the Witwatersrand. (The sputa were taken immediately after the diagnosis was made and before any treatment had been given.) Among the 134 specimens of sputa in the survey were several from cases of pneumonia in the series of 52 here under consideration. The prevalent bacterial flora associated with the pneumonia were demonstrated in an extract from Ordman's result of the survey:—

'Of all the cases investigated, 78.4% were associated with a pneumococcus, and in 21.6% of the cases clinically diagnosed as pneumonia, a pneumococcus was not discovered either by cultural or bacteriological examination. The *Micrococcus catarrhalis* and non-haemolytic streptococcus were isolated from nearly all of the specimens.

The following pathogenic respiratory organisms were recovered:—

A haemolytic streptococcus occurred in 25%, the *Haemophilus influenzae* in 40% and the *Staphylococcus aureus* in 4% of the specimens.

In brief, therefore, the pneumococcus types, 1, 2, 7, 14 and 17, together with a haemolytic streptococcus and a *Haemophilus influenzae* represented the bacterial flora.'

## RADIOLOGY

Radiographs were taken as soon after admission as convenient and subsequently sufficiently often, to establish the progress of the disease. The distribution of the lesions in the 52 cases was as follows:—

Site of Lesion	No. of Cases
Right base (including right midlobe) ... ..	20
Left base ... ..	13
Both bases ... ..	3
Right apex ... ..	8
Left apex ... ..	1
Diffuse ... ..	2
Bronchopneumonia ... ..	3
Right apical and midlobe together ... ..	2

This conforms with observations by others<sup>13,17</sup> that the right side is more commonly involved in bacterial pneumonia than the left.

The types of lesions on the radiographs were as follows:—

Classical lobar ... ..	4
Partial lobar ... ..	27
Segmental ... ..	12
Multiple segmental ... ..	2
Diffuse ... ..	2
Bronchopneumonia ... ..	3
Radiograph negative ... ..	2

1. *Classical Lobar Pneumonia*. Classical lobar pneumonia is a bacterial infection of sudden onset characterized by inflammation of the lungs, with, clinically, toxæmia of varying intensity, fever which usually terminates by crisis, raised pulse and respiratory rates, and rusty

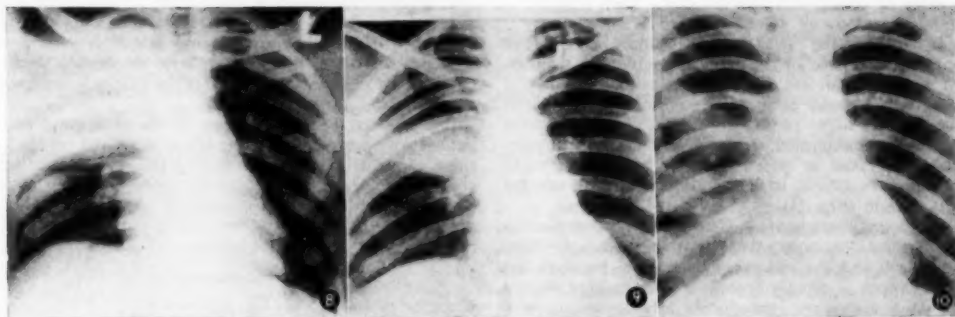


Fig. 8. Classical lobar pneumonia.

Fig. 9. Partial lobar pneumonia.

Fig. 10. Multiple segmental pneumonia, with 5 acute lesions in the lower part of the right lung field.

sputum.<sup>18</sup> The lung shows signs of diminished movement, dullness, bronchial breathing, crepitations and bronchophony over one or more lobes. Radiography of the chest shows opacity involving the whole of one or more lobes (Fig. 8).

2. *Partial Lobar Pneumonia.* This may present all the signs of classical lobar pneumonia, several adjacent broncho-pulmonary segments in one lung being involved. On radiography the shadow is frequently clearly demarcated on the one side by the inter-lobar septum (Fig. 9).

lung substance (Fig. 1B), both conforming in size and position, anatomically, to a broncho-pulmonary segment in the lung. Fig. 10 is a case of multiple segmental pneumonia showing 5 separate segmental areas of consolidation, all about the same size, in the lower half of the right lung.

The history is usually indicative of a chest complaint; either a definite localized pain, or a vague discomfort, or the chest may not be referred to at all in the history. The sputum may be frothy, mucous or bloody. Auscultation

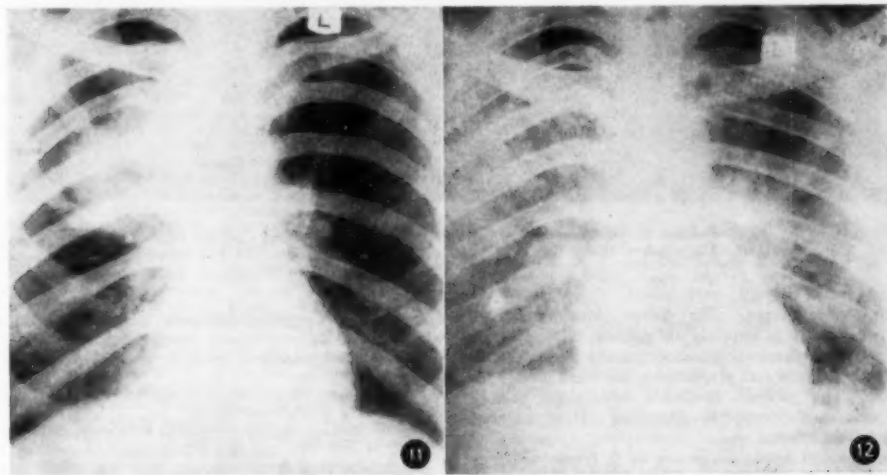


Fig. 11. Central pneumonia.

Fig. 12. Diffuse pneumonia.

3. *Segmental Pneumonia.* This is taken to be a fan-shaped shadow of consolidation with the base towards the periphery and the apex towards the hilum<sup>19</sup> (Fig. 1A), or a small circular area of consolidation, situated in the

may reveal only rhonchi which, if localized to one lobe, are suggestive of pneumonia in that lobe. Rhonchi, if generalized, in making one think along the lines of bronchitis, may mislead one into overlooking the signs

of a patch of pneumonic consolidation. Such a lesion, if centrally situated, may not be detected at all clinically.

Radiologically the size of the shadow corresponds roughly with the size of the broncho-pulmonary segment.

4. *Central Pneumonia.* This is characterized clinically by pyrexia, increased pulse and respiratory rates, bloody sputum and, as often as not, no clinical chest signs other than, perhaps, diminished air entry and localized rhonchi. Radiographs taken in postero-anterior and lateral views (the latter often difficult to read) indicate a shadow situated near the hilum (Fig. 11).

5. *Spreading Pneumonia.* Usually, after commencing Sulphapyridine treatment, the symptoms and signs show improvement and the radiographic shadow becomes less dense, although it usually spreads a little in area. Cases which do not improve immediately after treatment with Sulphapyridine are either those which spread for a few days and then begin to resolve, or those which continue to spread until some other form of chemotherapy is resorted to.

7. *Radiograph Negative Pneumonia.* These cases have pyrexia, increased pulse and respiration rates, bloody sputum, a definite leucocytosis and few, if any, physical signs, detectable in the chest and no discernible shadow develops on repeated radiograph if the case is treated with chemotherapy early.

Fig. 13A is the reproduction of the radiograph of a patient who presented with the above signs and no clinical signs of pneumonia in the chest. He was treated with chemotherapy immediately. After cure, Fig. 13B shows that a shadow had been missed, on the previous radiograph, extending down and merging imperceptibly with the right border of the heart shadow.

#### BRONCHOPNEUMONIA

Bronchopneumonia is an inflammation of the small bronchioles and the peribronchiolar alveoli.<sup>20</sup> The lobule is usually regarded as the unit involved by this inflammatory process.

Of 730 cases diagnosed as pneumonia, 26 (3.5%) were

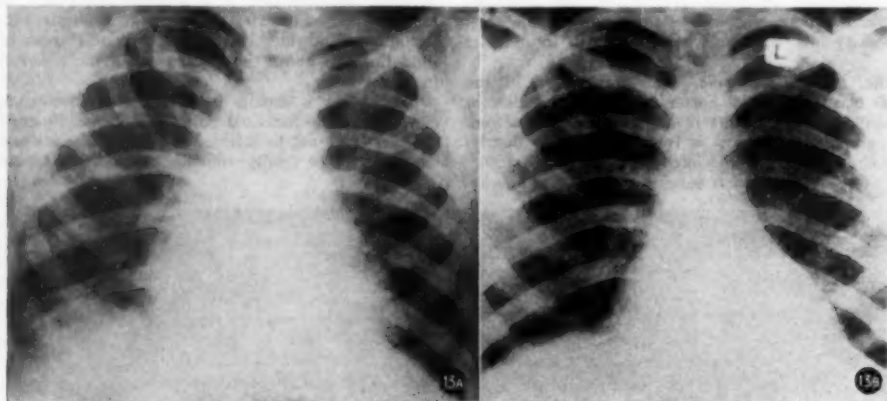


Fig. 13A. Shadow of pneumonic consolidation merging with the right border of the heart.

Fig. 13B. The shadow down the right border of the heart in Fig. 13A has disappeared.

6. *Diffuse Pneumonia.* The diffuse pneumonia case initially presents as a seriously ill patient, with pyrexia, raised pulse and respiration rates, usually with bloody sputum, with rhonchi and crepitations scattered over both lung fields. The patient's condition deteriorates rapidly and dullness and bronchial breathing develop later, involving whole lobes.

The radiological appearances are of 2 types—either a generalized uniform haziness throughout both lung fields, or small areas of consolidation a few millimetres in diameter, scattered uniformly throughout the lung substance against a background of uniform haziness. This whole picture is one of a rapid and widespread penetration of the organisms throughout both lungs, producing a fulminating disease (Fig. 12).

diagnosed as bronchopneumonia. This low incidence is what one would expect in a young adult population.

The bronchopneumonia pictures encountered fell into 3 groups:—

1. Acute bronchopneumonia (sub-divided into 4 types).
2. Creeping bronchopneumonia.
3. Recurrent bronchopneumonia associated with chronic respiratory infection.

1. Acute bronchopneumonia was the usual type of bronchopneumonia encountered. As in the types of pneumonia already discussed the pictures of acute bronchopneumonia are produced on the one hand by areas of infected atelectasis and on the other by the action of a more pneumotrophic organism on a sensitized patient.

(a) *Clinically acute lobular bronchopneumonia* (Fig. 14) manifests itself with pyrexia, sputum seldom bloodstained and

usually mucus or froth, with rhonchi and crepitations at the bases and with, perhaps, subsequently small areas of dullness and bronchial breathing.

The radiograph shows small patchy shadows stretching out from the hilar regions on both sides and extending towards both bases, obscuring the outline of the cardiac shadow and diaphragm, the patchy shadows being consistent in size with lobular involvement of the lung substance.

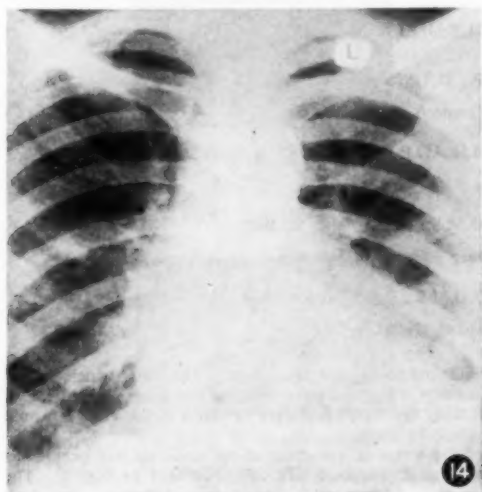


Fig. 14. Acute lobar bronchopneumonia.

(b) Clinically acute diffuse pneumonia shows a very toxic patient with high temperature, bloody sputum, generalized crepitations and rhonchi. Small areas of consolidation rapidly become confluent. In this type of case a virulent pneumotrophic organism effects a rapid, deep and widespread penetration of practically the whole lung substance, with widespread acute inflammatory congestion and oedema.

The radiographs show small irregular dense shadows scattered throughout both lung fields, set in a fairly homogeneous shadow of pulmonary congestion. This picture is indistinguishable from the diffuse pneumonia described above. The clinical signs, however, are those associated with a bronchopneumonia.

(c) Clinically bilateral, basal segmental, infected atelectasis presents initially pyrexia, mucus sputum and generalized rhonchi. Subsequently, at both bases, areas of dullness, bronchial breathing and a few crepitations come and go, with the sputum sometimes becoming bloodstained.

The radiograph shows bilateral fan-shaped shadows, identical with those seen in segmental pneumonia, resulting from infected atelectasis of a lung segment (Fig. 1).

(d) Clinically bilateral, multifocal, segmental pneumonia shows initially high pyrexia, rhonchi and crepitations at the bases, the sputum being bloody. Subsequently there is a bilateral dullness and bronchial breathing.

The radiograph shows circumscribed homogeneous shadows, initially usually 2 to 4 centimetres in diameter and situated in the lung substance isolated from the hilum. This appearance suggests an initial deep penetration into the lung substance by a pneumotrophic organism and the establishment of bilateral localized areas of inflammatory oedema. In Fig. 10, 5 such lesions are seen situated in the lower half of the right lung.

This brings the cases which clinically present a picture of acute bronchopneumonia, etiologically, clinically and

radiographically into line with the other types of acute bacterial infection of the lung substance described above.

2. The so-called 'creeping' bronchopneumonia is most frequently seen at the extremes of life and is consequently seldom encountered in the community here under consideration.

3. Recurrent pneumonia was most frequently associated with bronchiectasis.

#### SUMMARY

It is considered that the classification of bacterial pneumonia into lobar pneumonia and bronchopneumonia does not convey adequately to the clinician what is occurring in acute bacterial infection of the lungs and a classification is discussed to bring out the clinical forms which bacterial pneumonia may take.

Fifty-two cases of bacterial pneumonia are analyzed, the pathogenesis is discussed in regard to infected atelectasis and pneumotrophic organisms acting on the lung.

Average figures for pulse rates, respiration rates, white cell counts, duration of pyrexia, stay in hospital and working days lost are given.

It is shown that for the time intervals under consideration there is no correlation between the length of history, delay in the administration of Sulphapyridine, and duration of pyrexia.

The bacterial flora are analyzed and Sulphapyridine shown to be a satisfactory drug for treatment.

The clinical signs are correlated with the radiographical appearances.

I wish to thank General A. J. Orenstein and Dr. A. O. Dreosti, for their encouragement, criticism and permission to publish this paper.

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## TRACHOMA IN THE SOUTH AFRICAN BANTU A SURVEY IN SEKUKUNILAND

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As a result of recent surveys organized by the South African National Council for the Blind, it has been established that there are many cases of trichiasis, entropion and blindness among the South African Bantu in the Northern Transvaal. A 1949 survey in Sekukuniland reported a 22% incidence of trichiasis and entropion and a blind rate of 7.6 per 1,000 compared with less than 1 per 1,000 among South African whites.

To decide whether trachoma was implicated, a survey was made at the Jane Furse Memorial Hospital and surrounding districts of Sekukuniland approximately 200 miles north-east of Johannesburg. A total of 503 children ranging in age from 1 month to 18 years, of whom 281 were females and 222 were males, was examined. The first 114 came from Sekwati's kraal and the remaining 389 from Maulaneng.

The eye conditions in both villages and in both sexes were identical. We found naked-eye signs of infection with trachoma in 87% of the children. Only 68 cases (13%) had apparently normal lids. A random sample of 47 of these apparently normal children was examined by loupe, and 38 cases were found to have pannus—indicating that trachoma had healed without scarring but had left the diagnostic blood vessels in the cornea. These 38 cases raise the incidence to 94%. If the conditions of examination had been better, one feels the figure would be nearer 100%. This is what one might expect in view of the 100% incidence in the 3- and 4-year age groups.

Only 5 of 99 babies under 2 years escaped infection, while every one of 80 children aged 3 and 4 years was infected. Fig. 1a illustrates the typical picture of infiltrated palpebral conjunctiva in which the red velvety appearance was the feature (Stage 1 trachoma, i.e. Tr. I). The vascular pattern of the palpebral conjunctiva (Fig. 4) was lost in the early stages, which is in contrast to Egyptian trachoma. Less commonly a few yellow follicles were noted (Fig. 1b). Although the fly season had scarcely begun, many of the babies had clusters of

these insects around their eyes. The flies, which spread infection, can be discouraged by the simple expedient of washing the face. A supply of water in the villages would do untold good.

By 4 years of age most of the children had progressed to Stage 2 trachoma (Tr. II) illustrated in Fig. 2. The lids were seldom so thickened as to induce ptosis. Follicles could be seen in many cases. Others had healed without apparent scarring (Fig. 4), but examination by loupe revealed pannus. It was surprising to see scarring as early as 2 years (Fig. 3) and by 9 years scarring, Stage 3 trachoma (Tr. III), had developed in most lids. As scarring of the palpebral conjunctiva progresses, the upper lid develops entropion and the eye-lashes rub against the cornea (trichiasis). The cornea then becomes opaque and it is this fourth stage of the disease which causes so much blindness. Trichiasis, corneal scarring and pannus are illustrated in Fig. 6 (Tr. IV). This stage was seen in many parents but not in the children.

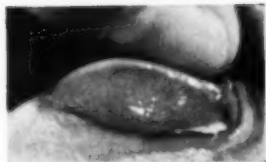
Follicles are sometimes present at the limbus corneae. When the follicles shrink they leave diagnostic facets, known as Herbert's pits (Fig. 7).

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Fig. 8 illustrates the frequency of the 3 stages in age groups. It can be seen that after 8 years of age, the active stages of the disease (Tr. I and Tr. II) rapidly diminish in number. Thereafter trachoma becomes more difficult to diagnose until scarring produces obvious entropion in later life.

*Halberstaedter-Prowazek Inclusions.* Although opinions are still divided about the nature of the causal agent of trachoma, some authorities accepting it as a virus while others regard it as riketsial, all are agreed that the presence of the Halberstaedter-Prowazek (H.P.) inclusion

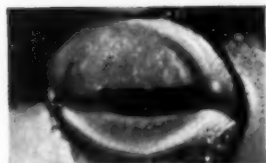




*Fig. 1 (a).* Tr. I. Infiltrative type.



*Fig. 1 (b).* Tr. I. Follicular type.



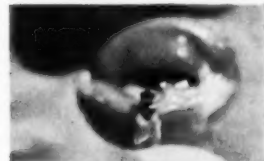
*Fig. 2.* Tr. II. Rough and thick.



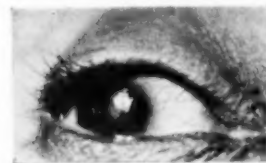
*Fig. 3.* Tr. III. Early scarring.



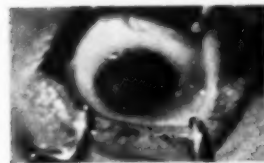
*Fig. 4.* Apparently normal, but cornea has pannus.



*Fig. 5.* Spreading the infection.



*Fig. 6.* Trichiasis and corneal scar.



*Fig. 7.* Herbert's pits at the limbus.

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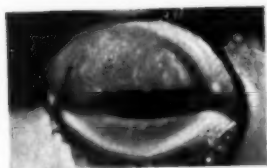
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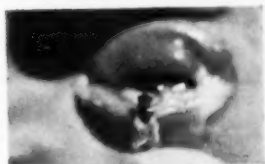
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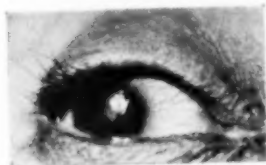
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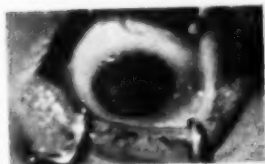
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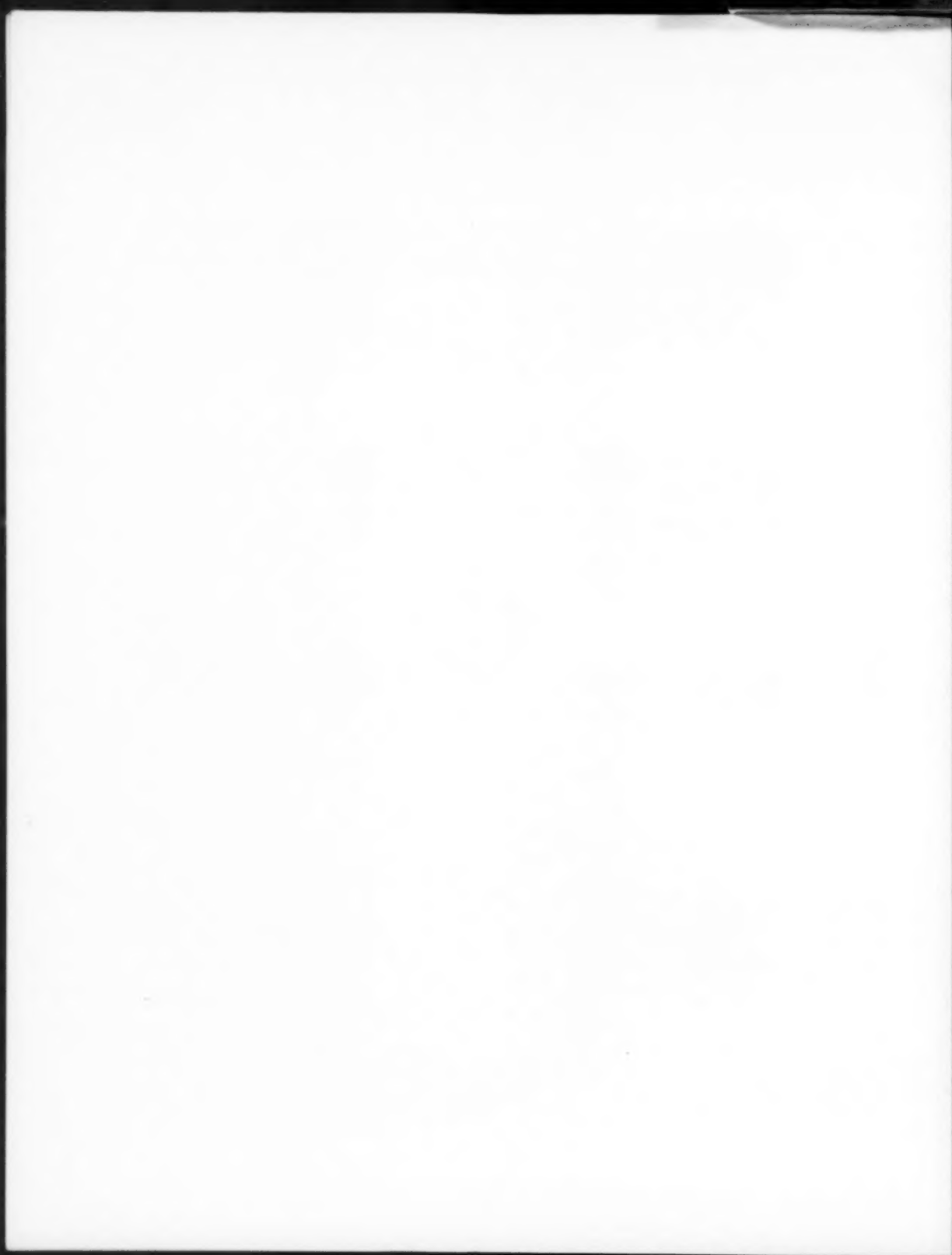
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bodies is, with one exception, pathognomonic of the disease. This one exception is the condition known as inclusion blennorrhoea of the newborn or swimming-bath conjunctivitis, in which intracytoplasmic inclusions are also found. This benign type of conjunctivitis is readily differentiated from trachoma on clinical grounds, since it does not cause pannus and leaves no scar tissue. It is important to note that H.P. bodies are never found in very large numbers in trachoma. During the first few weeks of infection they may be found readily, but as the disease progresses they rapidly decrease in number and are seldom discovered during the late stages.

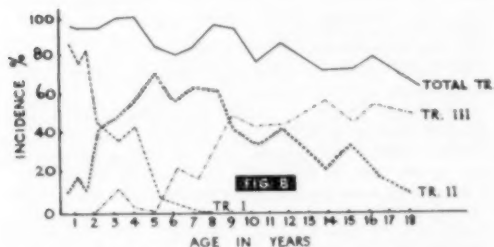


Fig. 8. Graph to show the incidence of Tr I, II and III in age groups.

Material for microscopic examination was obtained by lightly scraping the epithelium of the upper tarsal plate and superior fornix. The films were fixed immediately and stained by the long Giemsa method and by Gram. A total of 109 children assessed clinically as examples of Tr. I and Tr. II were examined in this manner. The results are summarized as follows:—

- Number of children examined: 109.
- With H.B. bodies: 25 (= 23%).
- With inflammatory exudate: 81 (= 74%).
- With Koch-Weeks infection: 70 (= 64%).
- With *H. Influenzae* infection: 12 (= 11%).
- With gonococcus infection: Nil.
- With pneumococcus infection: Nil.

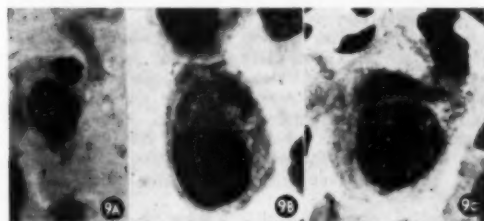


Fig. 9. Photomicrographs of intracellular inclusion bodies. (a) Morula Stage. (b) Mature Stage. (c) Rupture Stage.

The high incidence of Koch-Weeks (*H. conjunctivitis*) infection agrees with experience in other countries such as Egypt where trachoma is a common disease. The probable explanation is that the bacterial infection renders

the conjunctival epithelium more susceptible to invasion by the causal agent of trachoma. This concept of the pathogenesis has important implications with regard to the treatment of the disease, since Koch-Weeks infections respond well to sulphonamides.

The growth of the inclusion body from the morula stage to the rupture stage is illustrated in Fig. 9 A-C taken from 3 of our cases.

This demonstration of H.P. inclusions is of the utmost importance in proving our claim that the disease we were studying in Sekukuniland was, in fact, trachoma. In order to obtain independent confirmation of this, several of the stained conjunctival films were sent to Dr. F. Maxwell Lyons, Director of the Giza Memorial Ophthalmic Laboratory, Egypt, who kindly examined them and reported that he could, without hesitation, accept our findings.

#### DISCUSSION

It is well known that follicles occur in many types of conjunctivitis; that vascularity of the cornea is seen in some types of keratitis and in vitamin deficiency; that trichiasis and entropion result from such diseases as marginal blepharitis and pemphigus; but we claim that the findings in children of Stages I, II and III trachoma (each stage showing pannus), in an area where trichiasis and entropion are known to be rare, is clinical proof of trachoma. The findings of inclusion bodies is laboratory confirmation and makes irrefutable the diagnosis of trachoma in the South African Bantu.

It is probable that there are other areas where trachoma is less frequent or absent. It would be valuable if every doctor who has access to Native children would examine the everted lids of 100 children in the pre-school or early school age and report to the Bureau for the Prevention of Blindness, P.O. Box 1343, Pretoria. A map, showing the occurrence of trachoma throughout the Union, would be invaluable to the Public Health authorities in tackling this infectious, preventable and wasteful disease.

#### SUMMARY

1. Photographs of clinical trachoma and of inclusion bodies are presented to prove that trachoma exists amongst the South African Bantu.
2. Trachoma was found in 94% of the children in 2 villages near the Jane Furse Memorial Hospital in Sekukuniland.
3. Of the 503 children examined, 68 had apparently normal lids. When 47 of these cases were examined by loupe, 38 were found to have pannus. This indicates that trachoma heals not infrequently without scarring but leaves diagnostic blood vessels in the cornea.

#### ADDENDUM

The premise that trachoma would be found less commonly in other parts has been borne out by one of us (J. G. S.) examining 500 children near Mokhotlong in the highlands of Basutoland where no case of trachoma was found.

We are indebted to the South African Institute for Medical Research for a grant to cover the cost of the colour reproductions.

## ABSCESS TONSILLECTOMY

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A person suffering from a peritonsillar abscess is the personification of the term 'abject misery'. He can neither talk, eat, drink nor swallow without excruciating pain. So great is the pain that every attempt is made to avoid doing any of these things, as is shown by the continuous dribbling of saliva in the fully developed case. The patient's temperature is often well over 100° F and considerable toxæmia completes the picture.

A quinsy is a clinical rarity in the first decade of life and not often seen before 15 years of age. It is characteristically a disease of young adult life. Peritonsillar abscess can be defined as a suppurative process taking place in the loose connective tissue surrounding the capsule of the tonsil and separating it from the lateral pharyngeal wall. This layer of connective tissue is well developed in the upper two-thirds of the capsule of the tonsil but gradually fades away towards the lower pole. In the standard technique of tonsillar dissection the line of separation between the tonsil and the pharynx corresponds to this layer.

Statistics show that over 98% of quinsies occur in the upper pole.<sup>3</sup> Those occurring towards the lower pole are probably recurrent cases in which a previous abscess has obliterated the normal connective tissue layer.

The usual portal of entry of the infection to the peritonsillar tissue is by way of the tonsil.

The tonsils, in the case of quinsy, are almost invariably small. In our experience the tonsil is often not more than 1 inch thick and forms the medial wall of the abscess cavity.

It is usual to obtain a history of many severe attacks of tonsillitis. The onset of the quinsy is heralded by just one more attack of tonsillitis.

The characteristic clinical picture as outlined takes about 4-6 days to develop. There is no doubt that, if the condition is recognized early, it can be made to resolve rapidly by the use of antibiotics. Heavy dosage is essential and Penicillin in amounts of 600,000 units daily may be needed. If the antibiotic is successful, the patient will be markedly improved in 6 hours and symptom-free in 48 hours. Antibiotics cannot reasonably be expected to have an appreciable effect after the abscess is established.

The incidence of peritonsillar abscess is high. It was decided to collect the records of 25 consecutive cases. It took only 4 months at Grey's Hospital to accomplish this. During this period there were 12 cases which refused operation. From these figures it is reasonable to suppose that the incidence of the disease is about 100 cases per year at this hospital.

The treatment of quinsy has not changed for many years. It is barbarous in the extreme. The unfortunate patient is seated in a chair facing the well-intentioned doctor. The patient opens his mouth and a guarded knife or a special instrument reminiscent of the perforator of the dead infant's skull is thrust through the bulging anterior pillar into the abscess cavity. Pus is evacuated and, as with any

abscess, there is a marked improvement in symptoms. With the passage of time the patient recovers and returns to work. After such an episode, it is advised that the patient returns in 6-8 weeks for a tonsillectomy.

In the treatment of any disease the economic aspect should be considered. This falls into 3 parts:

- The loss of earnings to the patient.
- The cost of hospitalization.
- The loss of production of the patient's work.

The latter aspect, at times of emergency, might have serious results to the community as a whole.

The clinical course of quinsy after drainage by the approved method is seldom less than 14 days and it is doubtful whether the patient is fit to return to normal work in less than 21 days after the onset of the disease. At the end of 6 weeks it is proposed to admit the patient to hospital for removal of tonsils. Most adults are unable to work for 14 days after tonsillectomy. This condition of peritonsillar abscess will cause a loss of work amounting to 5 weeks under the normally accepted scheme of treatment. Much of this time may be hospital time, as it might be necessary to admit the patient to hospital because he has no place where he can be nursed adequately.

Abscess tonsillectomy<sup>1,2</sup> has been carried out in the past but has by no means been accepted widely as a feasible or desirable procedure.<sup>3</sup> It is defined as the operative removal of the tonsil when there is present a peritonsillar abscess, with the evacuation of the abscess. It is contended that by removal of the affected tonsil the widest drainage of the abscess area is produced. At least 45% of the abscess wall is removed.

Thompson and Negus state that they favour adequate drainage with later tonsillectomy and that they do not approve of abscess tonsillectomy. These two statements are difficult to reconcile, especially in view of the modification that must be made when one has adequate supplies of inexpensive antibiotics.

It was decided to embark upon a policy of abscess tonsillectomy at Grey's Hospital and the Out-Patient Medical Officers were instructed to admit every case of quinsy. The operation was performed, not as an emergency, but at the earliest convenient time. This was never longer than 24 hours after admission. Penicillin 100,000 units 6-hourly was started immediately on admission in every case and continued for three weeks after admission.

**Operation.** The anaesthetic was of the intra-tracheal type in every case. The tonsil on the affected side was removed without difficulty. The removal consists of dividing the attachment of the tonsil to the pillars and thus presented no difficulty. Bleeding was very slight and consisted of oozing from the divided pillars. All the vessels in the bed which usually give trouble in tonsillectomy are thrombosed. Packing of the cavity for 5 minutes is sufficient to arrest the bleeding.

In every case we have removed the tonsil on the other



side at the same operation. This has presented more difficulty because there have been adhesions and haemorrhage has been troublesome.

As a technical procedure we have found that it is far simpler to perform abscess tonsillectomy than to deal with the case of a healed quinsy, when the plane of separation is almost obliterated.

Post-operatively we were very surprised to find that the patients suffered very little pain and that a rise in temperature was the exception. The condition of the patient improved with great rapidity. It is our considered opinion that these patients recover very much more quickly than those of a similar age group whose tonsils are removed as a set operation.

On the average our patients left hospital on the fourth day fit and well. Some demanded to go home on the third day and as they appeared quite well and afebrile they were allowed to do so.

Healing of the operation site was satisfactory and rapid. No suggestion of any type of complication has been noted in any of the 25 cases recorded in Table I, or in any of the 32 subsequent cases treated similarly.

#### DISCUSSION

Any treatment of a disease which can materially shorten the period of absence from work without any increase in risk must receive due consideration from the profession. The routine treatment of quinsy involves a period of absence from work of about 5 weeks. Treatment of quinsy by abscess tonsillectomy has involved absence from work of less than 2 weeks in our series of cases. There is no undue difficulty in the performance of the abscess

tonsillectomy. It should be well within the capacity of any surgeon who has been in the habit of removing tonsils by dissection. Most of our later cases have been done by the Senior Resident Officers.

It is imperative that the anaesthetist should be completely competent and that intra-tracheal anaesthesia is the method of choice. In our experience the intubation of these patients has been the most difficult part of the whole operation. There is much swelling of the tissues of the pharynx, distortion of the normal anatomy and a gross excess of excretion.

There is no danger of spread of the infection as the most complete drainage of the abscess is obtained. Half of the wall, a hemisphere, of the abscess cavity is removed. There can be no rise in tension due to inadequate drainage and no tissue planes are opened. Pre- and post-operative antibiotics complete the safety of the procedure.

The shortening of the clinical course makes the treatment, from the patient's point of view, much more comfortable. When treated by the usual routine he suffers the opening of the abscess without adequate anaesthesia, the period of drainage and has the prospect of returning 6 weeks after recovery for a further operative procedure. Abscess tonsillectomy is done immediately under a general anaesthetic. It relieves completely all the symptoms. It is final and curative.

#### CONCLUSION

We recommend the treatment of peritonsillar abscess by the method of 'abscess tonsillectomy' as being a safe and relatively simple procedure.

TABLE I.

Case No.	Sex	Age	Lesion	Length of History	Post-Operative Course	Day of Discharge	Chemotherapy
1 .. .. .	F	22	Right Quinsy	8 days	Uneventful	4th	Yes
2 .. .. .	M	17	Left Quinsy	3 days	Uneventful	4th	Yes
3 .. .. .	M	35	Left Quinsy	9 days	Uneventful	4th	Yes
4 .. .. .	F	11	Right Quinsy	7 days	Uneventful	3rd	Yes
5 .. .. .	F	25	Left Quinsy	3 days	Uneventful	3rd	Yes
6 .. .. .	F	26	Right Quinsy	3 days	Uneventful	4th	Yes
7 .. .. .	M	24	Left Quinsy	10 days	Uneventful	3rd	Yes
8 .. .. .	F	16	Right Quinsy	5 days	Uneventful	3rd	Yes
9 .. .. .	F	34	Right Quinsy	2 days	Uneventful	4th	Yes
10 .. .. .	F	23	Left Quinsy	5 days	Uneventful	4th	Yes
11 .. .. .	M	22	Right Quinsy	4 days	Uneventful	4th	Yes
12 .. .. .	M	20	Right Quinsy	3 days	Uneventful	4th	No
13 .. .. .	F	25	Right Quinsy	4 days	Uneventful	4th	Yes
14 .. .. .	F	12	Left Quinsy	7 days	Uneventful	4th	Yes
15 .. .. .	M	25	Left Quinsy	3 days	Uneventful	3rd	Yes
16 .. .. .	F	20	Left Quinsy	7 days	Uneventful	7th	Yes
17 .. .. .	F	21	Right Quinsy	3 days	Uneventful	3rd	Yes
18 .. .. .	F	24	Left Quinsy	5 days	Uneventful	5th	Yes
19 .. .. .	F	55	Left Quinsy	7 days	Uneventful	7th	Yes
20 .. .. .	M	21	Right Quinsy	7 days	Uneventful	7th	Yes
21 .. .. .	F	19	Right Quinsy	6 days	Uneventful	4th	Yes
22 .. .. .	F	16	Left Quinsy	6 days	Uneventful	3rd	Yes
23 .. .. .	M	19	Left Quinsy	8 days	Uneventful	3rd	Yes
24 .. .. .	F	13	Left Quinsy	5 days	Uneventful	4th	Yes
*25 .. .. .	F	49	Left Quinsy	7 days	Fair amount of pain	7th	Yes

\* Tonsillectomy had been performed 20 years ago and the abscess was associated with a remnant.

Competent anaesthesia is of the greatest necessity and the use of antibiotics is desirable, both pre- and post-operatively.

Much hospitalization time is saved; the patient loses little work, and pain and suffering are reduced to a minimum.

#### SUMMARY

1. The pathology of quinsy is outlined.
2. The incidence of the disease is discussed.
3. Abscess tonsillectomy is defined.
4. The experience of 57 cases of abscess tonsillectomy

is presented. The detailed progress of 25 of these cases is tabulated.

5. A discussion on the merits of abscess tonsillectomy over the standard technique of treatment is given.

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## ELECTROSHOCK TREATMENT OF MENTAL ILLNESS DURING PREGNANCY

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The treatment of a psychotic episode, or a severe psychoneurotic reaction, arising during pregnancy, is a problem with which the psychiatrist is confronted not infrequently. In these cases the nature of the mental illness often provides a definite indication for the administration of electroshock treatment, usually, however, embarked upon with a good deal of trepidation and hesitation, because of the danger of interfering with the pregnancy.

The paucity of the literature on electroshock treatment during pregnancy is surprising. Kalinowsky and Hoch,<sup>1</sup> in their otherwise most comprehensive treatise, make only a very brief reference to two previously published articles, and offer no personal contribution to the problem.

In 1941 the first report emerged from Goldstein, Weinberg and Sankstone. They reported a single case where a series of 13 seizures was produced with Metrazol, the treatment being initiated during the fourth month of pregnancy. The gravid uterus had at first been misdiagnosed as a fibroid growth.

The patient was depressed and deluded, and the response to treatment was poor. A short course of sub-coma insulin was also given. This pregnancy proceeded normally, and a normal full-term infant was born. (It should be made clear that seizures produced by pharmaceutical preparations such as Metrazol are analogous to those produced by electrical means.) These writers also make mention of an observation by Clemmensen (1927) who reported on 107 pregnancies affecting 58 women. These were all epileptic patients, and in not a single case was there an abortion or interference with labour as a result of epileptic seizures.

Thorpe<sup>2</sup> administered 13 electroshock treatments to a young melancholic patient in her second pregnancy. Treatment started at the beginning of the sixth month and continued for six weeks. The mental condition resolved and the patient was delivered of a normal baby at full term.

Polatin and Hoch<sup>3</sup> recorded two further cases. In the first case electroshock treatment was begun during the fourth month of pregnancy. A depressive episode responded well to 6 treatments. Pregnancy proceeded normally and culminated in a normal delivery with the birth of a normal infant. The second case, a depressed

psychoneurotic, was given 10 electroshock treatments, commencing at the seventh month of pregnancy. The patient's depression later responded to psychotherapy. The pregnancy continued normally and a healthy infant was born at full term. The writers remark on the well-established fact that incontinence of urine or faeces during therapeutic seizures is rare. They suggest that electroshock has little influence on the smooth muscle of internal organs, and they therefore infer that it does not cause uterine contraction.

Simon<sup>4</sup> added 3 case reports. The first woman, suffering from an agitated depression, was given 6 electroshock treatments, commencing at the end of the third month of pregnancy. The infant was delivered at full term but died 2 days later. The author points out, however, that the patient had a 'stormy and toxic pregnancy'. The second woman, a psychoneurotic, was given 10 electroshock treatments early in pregnancy. She responded well, but later relapsed, and a further 4 treatments were given in the final stages of pregnancy. An uneventful delivery and the birth of a normal infant followed only 10 days after the last treatment. The third patient, agitated and depressed, was given 13 electroshock treatments in the latter half of pregnancy, the last one 29 days before the full-term delivery of a normal child. The latter two cases were of special interest as treatment was given in a very advanced stage of pregnancy.

Block<sup>5</sup> reports a case where treatment was continued into the final stages of pregnancy. Treatment was started at 5 months and continued intermittently throughout the remainder of pregnancy. The patient was psychotic. Thirty electroshock treatments in all were necessary and the last treatment was given one day before the full-term delivery of a normal child. A greater number of treatments was administered in this case than in any other reported.

Granlick<sup>7</sup> records a case which culminated in the delivery of a macerated foetus during the last month of pregnancy. From the end of the first month to the fifth month of pregnancy, this patient received 24 electroshock and 8 insulin-coma treatments. Foetal movements were felt one month after the conclusion of all treatment.

Three additional cases are reported by Kent.<sup>5</sup> Her first patient, aged 35, was a case of dementia praecox who had 2 courses of electroshock treatment before the reported pregnancy. This patient was given 3 electroshock treatments a week for 2 weeks, as well as insulin daily while she was ambulatory. She then delivered spontaneously a four-month foetus. Whether this abortion occurred immediately following the last treatment is not reported. Kent's second case was a manic patient. Here, during the sixth and seventh months of pregnancy, the patient had 26 electroshock seizures. A normal infant was finally delivered by Caesarean section. The reason for the operation was not recorded. The last case, suffering from dementia praecox, was given 20 electroshock treatments and 8 insulin-comas during the fifth to seventh months of pregnancy. The normal delivery of a healthy child finally ensued.

Careful scrutiny of the literature was not rewarded by any further relevant information from the English-speaking countries. A significant contribution must be added, however, from a French author.

Porot records 3 cases treated personally, the patients being North African Natives. The first woman, inert and retarded, was given 10 electroshock treatments early in pregnancy. The pregnancy proceeded without incident, and a full-term normal infant was ultimately delivered. The next case, an extremely agitated patient, was treated during the seventh month of pregnancy. After the third electroshock seizure, treatment was discontinued because 'a minute quantity of blood was noticed in the urine which could have been of vaginal origin'. Subsequently, an incidental phlebitis developed in the patient's leg, but the pregnancy continued until the normal delivery of a healthy child. The third patient required treatment for a state of melancholy. The first electroshock seizure was followed by a slight vaginal haemorrhage, but there was no danger of abortion. Three weeks later, treatments were continued until 12 electroshocks had been given. Additional therapy in this case included 23 insulin-comas, and anti-luetic arsenic and bismuth. The patient's mental condition improved, and a normal full-term infant was born. Porot also refers to a case recorded by other French authors. This particular patient was given 7 electroshocks during her third month of pregnancy. Following a street accident 8 days later, this patient aborted. The same authors, Porot points out, refer to 3 further unpublished cases known to them. These patients were treated with Cardiazol. Two of the cases were pathological pregnancies with abortion occurring; the third was treated during the eighth month of pregnancy without incident.

I am able to contribute a case report of a patient treated very recently at Valkenberg Hospital. The case is of special interest in that electroshock treatment was administered right up to the last day of pregnancy.

M. M., a Coloured female aged 28, was admitted on 17 September 1951. Before admission this patient had quite suddenly lost interest in her work as a domestic servant, become depressed and subsequently made a suicidal attempt. Physical examination revealed an advanced stage of pregnancy. Mentally the patient was depressed and retarded. She seldom spoke, and when she did, it was mainly to comment on her sinfulness and unworthiness. She also admitted to auditory hallucina-

tions. Her mental condition represented a psychotic episode for which electroshock treatment was indicated.

Dr. P. Massey was consulted for an obstetrical opinion. The pregnancy was considered to be entering its last month, and it was agreed that electroshock treatment was not contra-indicated. Treatment was instituted and administered three times a week. A muscular-relaxant was not used.

On 15 October 1951, the patient was given her ninth treatment. Treatment had proceeded that far without incident. Nine hours after the last treatment the patient went into labour. The labour was a normal one and a healthy infant weighing over 7 lb. was born. The patient's mental condition had, unfortunately, not responded well to treatment.

#### DISCUSSION

Many more cases must have been treated with electroshock during pregnancy, but these instances have not been reported. A survey of the available recorded evidence has been presented in some detail, and an attempt must now be made to interpret this evidence.

Kent's first case requires comment. This patient, aged 35, aborted a 4-month foetus, for which electroshock therapy could be blamed. Certain complicating factors may, however, have been present. No comment was made on the patient's general physical and metabolic condition. The case was apparently one of long-standing dementia praecox, a condition which is not infrequently associated with a considerable degree of deterioration in physical health. The fact that insulin was administered daily to the ambulatory patient strongly suggests, furthermore, that she was, in fact, probably in a poor state of physical health.

The patient reported by Granlick delivered a macerated foetus during the last month of her pregnancy. It is significant that electroshock treatment was concluded during the fifth month of pregnancy, and that foetal movements were felt one month later. In these circumstances, it seems hardly feasible to hold the electroshock treatment responsible for the unfortunate result.

The mother of the infant who died two days after its birth, Simon reports, had 'a stormy and toxic pregnancy'.

Porot's report of slight vaginal haemorrhages is not conclusive. The 2 cases to which he refers following the use of Cardiazol are also not significant, as both these pregnancies were pathological. His reference to the woman, who aborted as a result of a street accident, does not require further comment.

In Kent's case, therefore, lies the only reported instance where electroshock treatment during pregnancy may have been the cause of a mishap, viz. the abortion of a 4-month foetus. As this patient's state of general physical health was under suspicion, it might have contributed to the accident.

Turning to the favourable reports, one finds that there have been 12 cases treated with electroshock, one with Metrazol and one with Cardiazol, without any deleterious effect. This very small number of cases becomes rather more significant when it is appreciated that over 80 individual shock treatments were administered.

It might have been suspected that the initial and final

stages of pregnancy would represent periods during which the administration of electroshock would be particularly hazardous. The available evidence has, however, not supported such a view, as treatments have been carried out uneventfully at all stages of pregnancy.

The number of treatments which can be given safely to any individual case during pregnancy would also appear to be virtually limitless. In Block's case the patient was given as many as 30 electroshocks, and the pregnancy proceeded normally.

In order to complete this discussion on electroshock treatment during pregnancy, I was anxious to obtain the viewpoint of an obstetrician. Dr. P. Massey was approached, and her remarks are quoted:

'It is well known that epileptic seizures in the pregnant mother have no effect on the foetus, and at different times I have seen women delivered of normal infants following epileptic seizures during pregnancy. In one such case I recall, the woman had had over 80 major seizures.

'In eclampsia we always aim at reducing the number of seizures, as we believe that the fewer seizures that occur, the less danger there is to the foetus. Nevertheless, in cases of severe toxæmia without eclamptic seizures, the foetal mortality is often very high. It is generally accepted to-day that it is the toxæmia that is lethal to the foetus, and not the actual eclamptic seizures.

'The electroshock seizure differs little from epileptic and the eclamptic seizure, except that it appears to be, if anything, of slightly shorter duration. It is also significant that the period immediately following the seizure in which asphyxia can occur, is supervised, and resuscitation can be employed should it be necessary.

'It would seem that electroshock may be instituted during pregnancy with reasonable confidence. In addition to the literature quoted in this article, further case reports would be required to confirm this view.'

#### SUMMARY

A review of the literature of electroshock treatment during pregnancy has been presented. An additional case report

has been contributed. In all, 12 favourable reports have been recorded from the literature, over 80 individual treatments being involved. Treatment has been given at all stages of pregnancy. A single case has been given as many as 30 treatments. Of the unfavourable reports, there is only one in which electroshock treatment was possibly responsible for an abortion. In this case it was probable that there were other factors contributing to the abortion. In no instance did injury to a foetus occur which could be reasonably attributed to electroshock therapy. The personal views of an obstetrician, supporting the findings of this investigation, have been quoted.

#### CONCLUSION

The literature on the subject is very limited, and does not allow of any dogmatic statement about the risk involved in the administration of electroshock treatment during pregnancy.

The available evidence does suggest, however, that electroshock treatment of mental illness during pregnancy can be undertaken with the reasonable assurance that the pregnancy will proceed normally, and that the foetus will not be affected.

My thanks are due to Dr. G. Key, Physician-Superintendent, Valkenberg Hospital, for his permission to publish this article, and to Dr. P. Massey for her contribution and interest.

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##### PHLEBOGRAPHY IN NORMAL AND POST-PHLEBITIC STATES AND THE CLINICAL APPLICATION (MR. I. NORWICH)\*

The post-phlebitic syndrome is well known to all medical practitioners. It consists of bursting pain in the calf, especially on prolonged standing or at the end of a day, oedema, eczema, indurated cellulitis, varying degrees of ulceration and, in the occasional case, nocturnal cramps.

Different treatments have been tried to help the large number of patients affected. The treatment of the original deep thrombophlebitis is important. With the advent of anticoagulant therapy many more cases will be relieved completely or almost completely of these complications. However, a large group of patients do not receive anticoagulant therapy at the appropriate time or for a sufficiently long period, so that the post-phlebitic syndrome continues to be an important condition. There is also a group of cases with no antecedent history of

deep venous thrombosis. This group is classified as idiopathic and the etiology is considered to be a congenital incompetence of the valves of the deep venous system and comparable to that of superficial varicosities.

The many treatments of the established post-phlebitic syndrome that have been introduced are:—

1. Efficient and proper supportive bandaging.
2. Interruption of the sympathetic pathways to the affected limb.
3. Excision of diseased skin and grafting, in addition to surgery of the venous system.
4. More recently ligation of the deep venous system at the level of either the superficial femoral or the popliteal vein.

Ligation of the deep venous trunk is being employed, and results have been published by Linton, Bauer and others. The rationale appears to be based on the fact that, as the incompetent deep veins allow some blood to return through the

\*Paper read at a Meeting of the Association of Surgeons, Johannesburg.

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canalized thrombi, this is sufficient to prevent an efficient collateral venous return. Secondly the deep incompetent valves allow a reflux of venous blood which causes a venous hypertension especially in the erect position. Ligation of such a vein will stimulate the development of more efficient collaterals and also prevent reflux of venous blood on standing with all its associated complications. If one considers the pathology of the post-phlebotic syndrome to be a venous hypertension of the lower limb, then this method of dealing with it would appear to be logical.

In order to ascertain whether such deep veins are incompetent, retrograde phlebography is used. Phlebography of the deep venous system was originally carried out by means of the simple ascending technique of Mahorner. A light tourniquet is placed just above the ankle to occlude the superficial veins and injection of a dye is carried out into a foot vein with the patient supine. The retrograde method is not quite as simple. There are two techniques, the blind percutaneous and the cut-down. In this latter method, the femoral vein is exposed through a short vertical incision. After occluding the long saphenous and the common femoral vein proximal to the point of entrance of the needle into the deep femoral vein, the dye is injected and X-ray films are taken of the limb with the table tilted as far as possible so as to obtain an almost erect position of the patient.

**Clinical Significance of Retrograde Phlebography.** One must consider firstly the pathology of the well-established post-phlebotic syndrome. The original thrombus wherever situated organizes with scar tissue along the intima of the vein, and the pliability of the venous valves is interfered with causing deep vein incompetency against gravity. The condition is really a venous hypertension. Clinically this is borne out by the oedema and bursting character of the pain which is worse at the end of prolonged standing or at the end of a day's work. This is again substantiated by measurements of superficial venous pressures. In the normal standing individual the superficial venous pressure varies from 70 to 100 mm. Hg. With exercise it is immediately reduced to 0-30 mm. Hg due to the muscular pumping action of the lower limb plus the venous suction of the right side of the heart. In a post-phlebotic case, however, with prolonged standing or at the end of a day's work, the pressure will rise to well beyond 110 mm. Hg and with active exercise of the calf muscles the pressure remains between 50 to 110 mm. Hg because if the valves are incompetent reflux of blood takes place through to the superficial system and in spite of muscular exercise of the limb the pressure does not drop.

In order to appreciate the clinical significance of retrograde deep venous phlebography the number and site of the deep vein valves must be understood. One has dissected a number of these limbs in cadavers and a pattern determined. Powell and Lynn of the Post-Graduate Medical School, London, have recently dissected the limbs of 27 cadavers and concluded that great variations existed in different limbs of different and the same subjects. However, a workable pattern has been worked out by them.

In the external iliac vein, valves were present in 33%—1 only, occasionally 2.

In the common femoral vein, valves were present in 72%—only 1.

In the superficial femoral vein, valves were present in 100%—1-4.

In the upper two-thirds of the popliteal vein, valves were present in 96%—1 only, above the knee joint.

A great number of swollen painful limbs occur with ulceration in the distal leg in the Bantu. In the Non-European Section of Edendale Hospital we have had an opportunity of performing a number of retrograde phlebograms in these limbs which were clinically diagnosed as post-phlebotic states. An equal number of limbs with normal deep veins was investigated those on whom ordinary high ligations and phlebectomies were done for varicose veins.

Phlebograms were shown to the meeting.

It is interesting to note that the phlebograms conform to 3 patterns.

1. The dye is held up at the first competent valve, usually situated a short distance distal to the lesser trochanter of the femur. The dye is effectively held up and does not proceed distally. This picture presented in a number of normal limbs.

2. The dye held up partially but a thin fainter shadow was seen trickling through to the next valve or even proceeding distally as far as midcalf. This was found in the normal group

and also in the group clinically diagnosed as the post-phlebotic state.

3. The dye communicated via a very large number of collateral and perforator veins (which themselves appear varicose) with the skin and subcutaneous veins especially in the upper part of the thigh. Here the dye may or may not be held up at a high valve.

In all a total of 30 phlebograms were performed in normal and clinically incompetent deep venous systems.

From the results obtained it is difficult to assess by retrograde phlebogram alone that incompetence of the deep venous valves exists. In the normal limb some dye certainly does go beyond the first valve. Some radiologists would regard this as competence of venous valves.

The pressure of the injected dye may be sufficient to open the leaves of the valve to allow some of it to proceed distally. It may also be argued that as one injects the dye blood is returning to the heart, and for this reason there can never be a state where the valve is completely and effectively closed.

In the third pattern we could demonstrate a large number of incompetent collateral communications with the subcutaneous and skin veins. This may have an important bearing on the management of such cases. This requires further investigation.

We are completely satisfied that the retrograde phlebogram provides insufficient evidence of incompetency or otherwise of the deep venous valves. Our radiological findings appear to indicate that competency of these valves varies tremendously from person to person and that physiologically these valves are not the water-tight system previously supposed. It was therefore decided to correlate the radiological findings with venous pressures. The first series of venous pressures that we decided to investigate were those of the superficial system. As already indicated, with exercise the normal leg reduces its superficial venous pressure to a figure of 0-30 mm. Hg. With deep vein incompetency we discovered that the superficial venous pressure was never reduced below 50 mm. Hg, and the range was 50-110 mm. Hg. In some cases an actual rise occurred. In others where symptoms are less severe there is a slight drop in pressure with exercise of the limb.

In addition to the superficial pressures it was decided to attempt to obtain some information about the pressure in the popliteal vein.

If a state of venous hypertension exists with incompetent deep veins then measurement of the pressure in the popliteal vein in the erect and horizontal positions should provide useful information. These pressures were obtained with the ordinary glass spinal manometer. The blood column itself was measured so that the pressures were recorded in mm. of blood. The apparently normal popliteal vein pressure was measured at the time of ligating the lesser saphenous for the operation for varicose veins. In this series a number of so-called normal readings were obtained. In another series, popliteal pressures were measured in a series of the post-phlebotic state. The readings were taken with the patient horizontal on the operating table, and then, with the table tilted as near the vertical position as possible.

A few of the results obtained are tabulated:

	Horizontal	Tilted
Case 1. Deep venous thrombosis ...	270	410
Case 2. Deep venous thrombosis ...	100	400
Case 3. Deep venous thrombosis ...	60	420
Case 4. Varicose veins, one side ...	140	495
Case 5. Varicose veins:		
Right ...	70	400
Left ...	115	550

From these few figures it is obvious that this investigation is of no real value. Incompetent tributaries of the popliteal vein may upset the results. This is disappointing because one had hoped that this investigation would be of value in correlating the X-ray findings in order to select cases which would benefit from deep venous ligation.

A series of deep veins was interrupted for the established post-phlebotic syndrome. The popliteal vein was selected for the following reasons:

1. Venous communications from the profunda vein to the popliteal may open up the latter if this is ligated high. These venous communications are often visualized on retrograde phlebography.

2. Phlebographic studies taken 11 and 3 minutes after injection of the dye show stasis in the main venous channel

and indicate high pressure in the popliteal vein. Ligation of the popliteal vein would therefore relieve this.

3. Pronounced stasis phenomena seldom occur in the thigh. Good collaterals exist in the thigh region because of abundant muscular veins. The leg however has much poorer opportunities in this respect because the blood return must pass through a bottle-neck at the level of the knee joint, via the popliteal and long saphenous. If the popliteal is ligated blood is forced through the saphenous if it still exists and through the capsular (articular) veins into the thigh and once into the thigh the return flow is adequately taken care of.

So far a series of 35 popliteal ligations have been carried out for the established post-phlebotic syndrome. Most of these have been investigated by retrograde phlebography. The results so far after a follow-up of about 2 years are as follows:

**Ulceration and Eczema.** Approximately 65% showed signs of satisfactory improvements but the rest had recurrences. The typical indurated cellulitis also showed improvement.

**Bursting Pain.** Improvement of this symptom was noted in approximately 80% of cases. This is especially interesting because the oedema showed no improvement.

**Oedema.** Not improved and any improvement that did result was possibly due to the efficient supportive bandage or elastic stocking worn post-operatively.

**Nocturnal Cramp.** This occurred only in 2 of these series and showed definite signs of improvement after popliteal ligation.

In summarizing these incomplete remarks it may be said that, with this limited number of investigations and cases:

1. The prophylaxis of the post-phlebotic state with the use of intensive anticoagulants in the early acute phase is most important.

2. Retrograde phlebography is no sure means of estimating

deep vein incompetence. It is merely an investigation complementary to the clinical findings.

3. Superficial venous pressures are of great value and it is possible that the selection of cases for popliteal ligation will be based on the variations determined in the superficial venous pressures.

4. Pressures of the popliteal vein as described here are of no value in estimating the degree of venous hypertension. It is possible that some other means could be devised to carry out this investigation. Venous haemodynamics needs further investigation.

5. Popliteal vein ligation for the post-phlebotic syndrome has a place in selected cases. The indications will be established with experience and further investigation. Some of the disabling symptoms show improvement in a percentage of cases but not in all.

6. One group of retrograde phlebograms illustrates a large number of what appear to be incompetent perforators and collaterals. This requires further investigation because, without surgical attack in these, popliteal and femoral vein ligation alone may be insufficient to relieve venous hypertension completely.

## PASSING EVENTS

Mr. H. Katz has returned to Cape Town from London where he has been doing post-graduate surgical study.

### CAPE TOWN PAEDIATRIC GROUP

At the next meeting to be held on 2 May in the Little Lecture Theatre, Groote Schuur Hospital, at 8.15 p.m., Dr. S. Scher will speak on *Renal Infections in Childhood*.

## CONGRESS 1952

### SOUTH AFRICAN MEDICAL CONGRESS 1952

The Honorary Organizing Secretary of the Congress has been advised that the replies on the Intention Cards have been somewhat vague. The Publicity and Travel Department of the S.A. Railways is anxious to have the following information as soon as possible:

(a) Whether suite, room with private bath or ordinary room is required.

(b) If unaccompanied, whether prepared to share a double room.

(c) Mode of travel to and from Johannesburg and whether bookings, rail, air or road-motor required through the S.A.R. Publicity and Travel Department.

(d) Date of arrival at and departure from Johannesburg. Intending visitors to the Congress should communicate immediately with the Honorary Organizing Secretary at Medical House, 5 Esselen Street, Hospital Hill, Johannesburg.

## REVIEWS OF BOOKS

### INDUSTRIAL MEDICINE

*The Application of Scientific Methods to Industrial and Service Medicine.* (Proceedings of a Conference held from 29 March to 31 March 1950.) Medical Research Council. (Pp. 112, with figures. 3s.) London: His Majesty's Stationery Office. 1951.

**Contents:** 1. Opening Address: H. P. Himsworth. 2. Scientific Method in Field Surveys. 3. The Investigation of Health Hazards: I. 4. The Improvement of Human Performance by Laboratory Studies: I. 5. The Improvement of Human Performance by Laboratory Studies: II. 6. The Investigation of Health Hazards: II. 7. Closing Session: Lessons of the Conference.

The papers dealt with at the above Conference may be divided into 3 main groups, the first being the *Scientific Methods in Field Surveys*, in which Professors Bradford Hill, Squire and their associates exalted common sense into a science and stressed the pitfalls of the statistical approach to those problems affecting both industrial and service medicine requiring answers. The importance of planning any statistical survey, with emphasis on sampling and controls, were emphasized by all.

The second group of papers dealt with the *Scientific Method of Investigating Specific Industrial Hazards* in which Dr. Donald Hunter's paper on fluorosis and berylliosis is a model of all that is at present known of these compounds as they affect the industrial worker.

The third group dealing with the improvement of the *Performance of Individuals as Elicited by Laboratory Studies*

must take precedence in so far as Industrial and Service Medical Officers are concerned with particular emphasis on the excellent papers by Drs. Bedford and Weiner. Both papers should receive careful study by those medical personnel charged with the health of those engaged in our main industry—the Gold Mines.

It is quite impossible to do full justice in such a short review to the many excellent papers delivered at this Conference and it cannot be sufficiently stressed that this small volume should assume a prominent place on the library shelves of all concerned with Industrial and Service Medicine.

### FOR THE DOCTOR'S LEISURE

*The Quiet Art—A Doctor's Anthology.* Compiled by Dr. Robert Coope. (Pp. 284. 12s. 6d.) Edinburgh and London: E. & S. Livingstone Limited. 1952.

**Contents:** 1. The Science and the Art. 2. Students All. 3. Vignettes. 4. The Doctor's Workshop. 5. This Insubstantial Pageant. 6. The Compleat Doctor. 7. At the Close of the Day. 8. 'This Sanctuary of my Soul'. Index.

Dr. Robert Coope, well known for his excellent book on *Diseases of the Chest*, has taken time off from his more technical pursuits to produce an anthology for medical practitioners.

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## CORRESPONDENCE

## PSYCHOLOGICAL TREATMENT TECHNIQUE

To the Editor: A whisper or two of discontent from a medical practitioner or three concerning the views set out in my letter published in the *Journal* of 8 March 1952 has been brought to my attention.

Such discontent, I gathered, came from surgeons and physicians and not from psychiatrists, though I have no doubt that certain psychiatric groups, notably those stemming from the Church of Freud, would mutter darkly into their beards (if any) and murmur disgustedly of 'retrogressive psychological views' and 'archaic psychiatry'.

Let it be thought by any general medical practitioner reader of the *Journal* that I have perhaps been setting myself up as a voice crying in the wilderness of outmoded pre-scientific illusion, permit me to draw on 2 publications by Dr. William S. Sadler, Consulting Psychiatrist to Columbus Hospital and Fellow of the American Psychiatric Association.

In his book *The Truth About Mind Cure*,<sup>1</sup> first published in Great Britain in 1929, he wrote: 'I would, therefore, say this in closing this discussion on mind cure: since fear is the foundation of nerves and since faith is the only known cure for fear, and since religious faith is the master-mind cure, I would therefore make this recommendation—that we call a halt in this business of taking a page out of psychology to start a new religion; taking the heart out of the teachings of Jesus Christ to start a commercial system of religious mind cure; and, further, . . . that we call you people back to your long-neglected Bibles with their "exceeding great and precious promises", wherein it is written: "Cast all your care upon Him, for He careth for you"; "Come unto Me all ye that are weary and heavy laden, and I will give you rest"; and that "He healeth all our diseases"; and that you get a new vision of the carpenter's Son, going up and down this world two thousand years ago healing the sick and comforting the afflicted.'

If you are in need of a mind cure, if you are looking for a religious mind cure, turn not to some strange god or new-fangled modern cult; but if you need the Balm of Gilead for your soul and spiritual consolation for your mind, get a religion that will not only heal your body just as well as any of these new-fangled cults, but get a religion that also promises to do something for your soul, over in the great beyond, in the sweet by and by, where you need no more healing for the body. And that religion, in my opinion, is not some new and modern psychic phantasma, but the simple, old-fashioned Gospel of the Lord, Jesus Christ.'

Dr. Sadler also stated in that same volume: 'I would not have the reader believe that I think that the religions of pagan races and heathen lands are just as good as the Christian religion. I personally am a believer in the Christian religion, even though there are many things about it I cannot fully understand. Nevertheless, I reckon myself a believer in the fundamental teachings of Christ.'

One might perhaps expect that later developments in psychiatry might have caused Dr. Sadler to deviate from these views. Yet, in 1945, we find him publishing his 896-page textbook, *Modern Psychiatry*,<sup>2</sup> in which, integrated with numerous sound observations that take into account contemporary scientific findings, he deviates not at all from his views published some 20 years earlier.

Far from Dr. Sadler's being a lone voice crying unscientifically in an American wilderness, one may turn to a publication such as that of Dr. James D. Page, Associate Professor of Psychiatry and Director of the Psychological Clinic of Temple University, U.S.A., who in his book *Abnormal Psychology*<sup>3</sup> states under the heading of *The Prevention of Crime*: 'Parents may discourage antisocial tendencies in their children by maintaining a favorable emotional atmosphere in the home, establishing friendly relations with their children, and carefully guiding their moral and character development. In the latter task, the church, the school, and the Scout movement may also play important roles.'

The old Scots Presbyterians had the right idea when they attempted to give to their children that priceless gem of

wisdom: 'Let us hear the conclusion of the whole matter: Fear God, and keep His Commandments; for this is the whole duty of man.'<sup>4</sup>

## REFERENCES

1. Sadler, William S. (1929): *The Truth About Mind Cure*, pp. 204-206. London: George Allen & Unwin Ltd.
2. Sadler, William S. (1945): *Modern Psychiatry*. St. Louis: The C. V. Mosby Co.
3. Page, James D. (1947): *Abnormal Psychology*, 1st ed., p. 408. New York and London: McGraw-Hill Book Co. Inc.
4. Solomon, King of Israel: *The Holy Bible (A.V.)*, Ecclesiastes 12: 13. Oxford University Press.

J. J. de Villiers, M.B., Ch.B. (Edin.)

224 Brook Street,  
Brooklyn,  
Pretoria,  
19 March 1952.

## MISTAKEN IDENTITY: A CORRECTION

To the Editor: It has been brought to my notice that the writings of a certain C. Valkenberg de Villiers, a non-medical quack of 4 Layton Mansions, 257 Jacob Mare Street, Pretoria, have been attributed by various medical practitioners and members of the general public to myself.

These writings, of some of which I have obtained copies, are defamatory towards various named registered medical practitioners and Government officials, as well as being filthy and blasphemous, and are circulated to the public in the form of roneoed 'open letters'.

Further, information was given me by a near relative some 6 weeks ago that he had received a letter from the Private Secretary to the Honourable the Minister of Health enclosing certain of C. Valkenberg de Villiers' roneoed material, and stating, as I myself noted in the letter, as follows: 'Dr. Bremer het my versoek om u aandag te vestig op die meegaande omsendbrief wat hom onlangs bereik het en wat blykbaar deur dr. de Villiers uitgereik is.' The Minister was promptly notified of his error, and the Registrar of the S.A. Medical and Dental Council was interviewed for his advice whether a disclaimer in the public press would constitute advertising and thus an infringement of medical ethics.

Mr. W. Impey's advice was that the publication of a disclaimer in the public press would constitute such an infringement, and could give no suggestion about how the Council could be of assistance in removing the erroneous impression from the minds of affected members of the public and of the medical profession.

It has therefore occurred to me that rectification of the position may be partially effected should this letter to you be published in the *Journal* or, alternatively, should it be passed on to the President of the South African Medical Association for consideration by the Executive Committee.

Needless to say the error in identity has operated injuriously in a number of ways, and the correction of a mistake such as this, which has penetrated even into Union Cabinet circles, is urgently required.

J. J. de Villiers, M.B., Ch.B. (Edin.)

224 Brook Street,  
Brooklyn,  
Pretoria,  
20 March 1952.

## TEAM-WORK IN DIABETES

To the Editor: Co-operation between doctor and patient is never more important than it is in diabetes. Being a chronic condition, it is more than a medical problem and also involves questions of personal and social adjustment which can often be handled more efficiently by a group than by individual doctors and patients.



In their efforts to cope with their various problems, diabetics often consult amongst themselves, they go to other amateurs, or to herbalists, quacks and others who may do more harm than good, and who often complicate the medical control of the patient.

The problem would become easier if the individual therapy of the doctor could be supplemented by a medically recognized group-therapy. Such therapy, aided by lectures given by medical men and recognized dieticians, and by publications of interest to both patients and doctors, is part of the work of the various Diabetes Associations which are already functioning in 13 countries, viz.: Australia, Belgium, Canada, Denmark, England, Finland, France, Italy, Netherlands, Portugal, Spain, Sweden and the U.S.A.

Since such organizations are under medical control, they do not compete but co-operate with the doctors who, like the patients, appreciate the research and the information originating from the work of these bodies.

Apart from their scientific work, these Associations tackle some of the material and psychological stresses facing the diabetic. They have already been successful in arranging camps for diabetic children, improving eating facilities for diabetics of all ages, arranging for the insurance of diabetic lives and facilitating the employment of diabetics in private and public enterprises.

High-grade diabetic necessities and luxuries are often submitted to the official investigation of such an organization, which then separates the wheat from the chaff and thus saves its diabetic members from unnecessary danger or expense.

Since the basic aim of all National Diabetes Associations concerns the legitimate interests of the diabetic, they federated in 1950, forming the International Diabetes Federation or I.D.F. Its honorary presidents are: Elliot P. Joslin of Boston and C. H. Best of Toronto. Another well-known authority is the President of the Executive Council, namely R. D. Lawrence, of London. The headquarters of the I.D.F. are at 33 Prinsegracht, The Hague, Netherlands, which is also the address of its Secretary-Treasurer, Dr. F. Gerritzen.

It is also in the Netherlands that this year's International Congress on Diabetes is being held under the auspices of the I.D.F. and we hope that more National Diabetes Associations, including a South African one, will gradually come into being.

If this letter is read by anyone interested in the establishment of a South African Diabetes Association, further details may be obtained from the undersigned (Dr. R. Schweitzer). We would also be interested in comments and suggestions on this matter.

T. Schneider, M.B., B.Ch. (Rand), R. Schweitzer, M.B., Ch.B.,  
M.R.C.P. (Edin.), P.O. Box 171,  
Queenstown, C.P.

Diabetic Clinic,  
Johannesburg Hospital,  
Johannesburg.

31 March 1952

#### PROF. CILLIERS' MINERVA CALENDAR

To the Editor: May I congratulate you on your very excellent editorial in this *Journal* on 19 January, in which you discussed the Minerva Calendar and its use in family spacing.

Prof. A. C. Cilliers deserves the thanks of the profession for offering them an appliance which can be recommended with confidence to their patients. Here is no haphazard fame-seeking gadget, but a neat invention, carefully designed and based upon the widely accepted Ogino-Knaus theory of reproduction. Prof. Cilliers also deserves credit for offering his invention to the public via the Medical Profession.

As a urologist I am particularly interested in the Calendar for those couples who find difficulty with conception. In addition to any treatment given to husband or wife, it is mandatory for them to know of the 'fertile days'.

Our best means of establishing the time of ovulation accurately is by taking the early morning rectal temperature. As this knowledge is most important, whether one seeks fertilization or to avoid it, I would like to suggest to Prof. Cilliers that he adds a temperature chart to his calendar strip.

There seems to be space enough for it, and I am sure my gynaecological colleagues will agree with me that it would add considerably to the versatility of the appliance.

P. J. M. Retief.

514 African Life Buildings,  
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31 March 1952.

#### MEDICAL PRACTITIONERS AND INDUSTRIAL LEGISLATION

To the Editor: With reference to the letter from the Assistant Medical Secretary dated 4 March 1952, and the recent telephonic conversation you had with an official of this Department, I have to advise you that all medical practitioners who are employers of labour are required:

(a) To register in terms of the Unemployment Insurance Act, No. 53 of 1946, as amended. In this connexion they are kindly requested to communicate direct with the Unemployment Insurance Commissioner, P.O. Box 1052, Johannesburg.

(b) To register in terms of the Workmen's Compensation Act, No. 30 of 1941, and in this connexion they are also required to communicate direct with the Workmen's Compensation Commissioner, P.O. Box 955, Pretoria.

In terms of Section 97 of the above Act they are required to keep a record of the wages paid and the daily hours of work of their employees.

(c) To pay their employees the prescribed cost-of-living allowances in terms of War Measure No. 43 of 1942, as amended. In terms of Regulation 3 (2) of the above measure they are also required to show the ordinary weekly or monthly remuneration separately from the cost-of-living allowance paid to each of their employees.

For your information the schedule of the cost-of-living allowances payable from 21 April 1952, is as follows:—

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" 5 8 4—6 10 0	2 14 2
" 6 10 0—7 11 8	3 6 1
" 7 11 8—8 13 4	3 11 6
" 8 13 4—9 15 0	4 1 3
" 9 15 0—10 16 8	4 6 8
" 10 16 8—11 18 4	4 16 5
" 11 18 4—13 0 0	5 6 2
" 13 0 0—14 1 8	5 17 0
" 14 1 8—15 3 4	6 6 9
" 15 3 4—16 5 0	6 18 8
" 16 5 0—17 6 8	7 9 6
" 17 6 8—19 10 0	8 3 7
" 19 10 0—21 13 4	8 17 8
" 21 13 4—23 16 8	9 10 8
" 23 16 8—26 0 0	10 5 10
" 26 0 0—28 3 4	11 1 0
" 28 3 4	12 0 6

If the ordinary weekly remuneration, plus cost-of-living allowance payable thereon, to any employee in terms of this Regulation, is at a rate which exceeds £18 per week, the weekly allowance payable to such employee shall be an amount equal to the difference between the said remuneration and £18. Any employee whose ordinary weekly remuneration is in excess of £18 is not entitled to a cost-of-living allowance.

\* C.O.L.A. means Cost-of-Living Allowance.

Note: Cost-of-living allowance must be reflected in a separate column in all Wage Registers.

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<sup>1</sup> Towse, R.C.; Berberian, D.A.; and Dennis, E.W.; *New York State Jour. Med.*: 50:2035, Sept. 1950.

<sup>2</sup> Berberian, D.A.; Dennis, E.W.; and Pipkin, C.A.; *Am. Jour. Trop. Med.*: 30:613, Sept. 1950.

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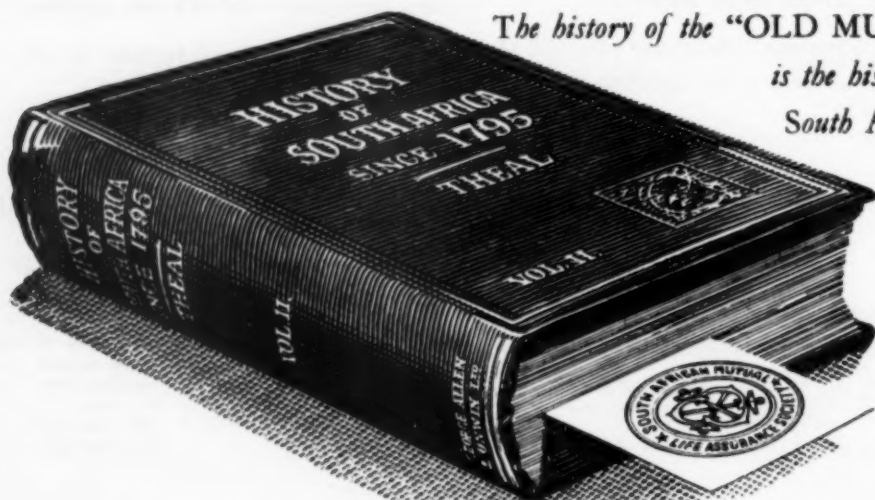


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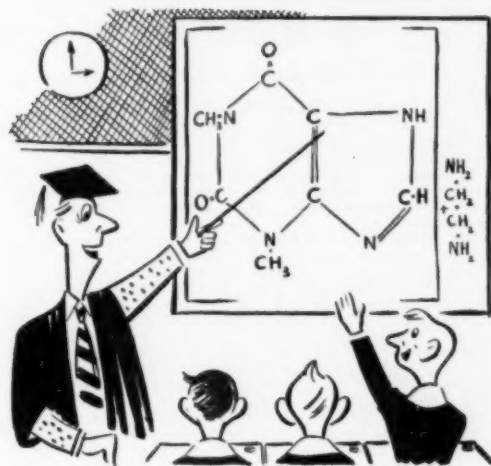


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P.18

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(981) Cape Town Northern Suburb. Assistant required for general practice. Salary to be arranged.

(960) Eastern Province hospital town. As soon as possible, an assistant with view to partnership. Car essential but would not normally be required for practice. Single man preferred but not essential. Remuneration to be arranged.

(887) Cape Town. Assistant required for approximately 1 year. Salary subject to mutual agreement.

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(811) Protestant G.P., qualified 9 years, married, with varied G.P. experience, seeks partnership.

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Die suksesvolle applikant moet op Port Elizabeth woon, op 'n datum wat gereël sal word diens aanvaar, en sy pligte ooreenkomstig die regulasies van die Siekfonds uitvoer.

Aansoeke moet die Distrikssekretaris, Distriksiekfondsraad, Kamer 116, Mutual-geboue, Hoofstraat, Port Elizabeth, nie later nie as 30 Mei 1952 bereik, en applikante moet die volgende vermeld:

1. Volle naam.
2. Kwalifikasies (waar en wanneer verkry en opgedoen).
3. Ondervinding (waar en wanneer verkry en opgedoen).
4. Datum van geboorte.
5. Land van geboorte.
6. Getroud of ongetroud.
7. Of ten volle tweetalig.
8. Of Suid-Afrikaanse burger.
9. Watter staatsbetrekking, indien enige, beklee word.

Werwing deur of ten behoeve van enige applikant stel so 'n applikant bloot aan diskwalifikasie.

Enige verder besonderhede wat verlang word kan op aanvraag van die Distrikssekretaris by die bovermelde adres verkry word.

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Applications should be submitted (in duplicate) on the prescribed form Staff 23, which is obtainable from the Director of Hospital Services, P.O. Box 2060, Provincial Building, Wale Street, Cape Town, or from the Branch Representative, of the Hospital Department at Cape Town (P.O. Box 1487), Port Elizabeth (P.O. Box 80), East London (P.O. Box 13), Kimberley (P.O. Box 618), and Umtata (P.O. Box 202), or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.

The closing date for the receipt of applications is 3 May 1952 and applications should be addressed to the Branch Representative, Hospitals Department, P.O. Box 1487, Cape Town.

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